INFORMATION TO USERS

This manuscript has been reproduced from the microfilm master. UMI films the text directly from the original or copy submitted. Thus, some thesis and dissertation copies are in typewriter face, while others may be from any type of computer printer.

The quality of this reproduction is dependent upon the quality of the copy submitted. Broken or indistinct print, colored or poor quality illustrations and photographs, print bleedthrough, substandard margins, and improper alignment can adversely affect reproduction.

In the unlikely event that the author did not send UMI a complete manuscript and there are missing pages, these will be noted. Also, if unauthorized copyright material had to be removed, a note will indicate the deletion.

Oversize materials (e.g., maps, drawings, charts) are reproduced by sectioning the original, beginning at the upper left-hand corner and continuing from left to right in equal sections with small overlaps.

Photographs included in the original manuscript have been reproduced xerographically in this copy. Higher quality 6" x 9" black and white photographic prints are available for any photographs or illustrations appearing in this copy for an additional charge. Contact UMI directly to order.

Bell & Howell Information and Learning
300 North Zeeb Road, Ann Arbor, MI 48106-1346 USA
800-521-0600

ATTENTION: HOW DOES IT MOVE US?
A KINEMATIC ANALYSIS OF THE EFFECTS OF VISUAL ATTENTION AND MOTOR PROGRAMMING ON MANUAL AIMING MOVEMENTS IN HEALTHY YOUNG AND HEALTHY ELDERLY INDIVIDUALS, AND THOSE WITH ALZHEIMER'S AND PARKINSON'S DISEASES

by

Susan Agnes Morehouse

Department of Psychology

Submitted in partial fulfilment
of the requirements for the degree of
Doctor of Philosophy

Faculty of Graduate Studies
Dalhousie University
Halifax, Nova Scotia
August 1999

DALHOUSIE UNIVERSITY

FACULTY OF GRADUATE STUDIES

The undersigned hereby certify that they have read and recommend to the Faculty of Graduate Studies for acceptance a thesis entitled "Attention: How Does it Move Us? A Kinematic Analysis of the Effects of Visual Attention and Motor Programming on Manual Aiming Movements in Healthy Young and Healthy Elderly Individuals, and those with Alzheimer's and Parkinson's Diseases"

by Susan Morehouse

in partial fulfillment of the requirements for the degree of Doctor of Philosophy.

Dated: August 9, 1999

External Examiner

Research Supervisor

Examining Committee

DALHOUSIE UNIVERSITY

DATE: August 28, 1999

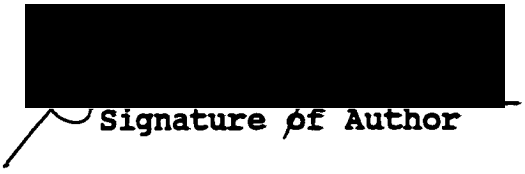
AUTHOR: Susan Agnes Morehouse

TITLE: Attention: How Does It Move Us? A Kinematic Analysis of the Effects of Visual Attention and Motor Programming on Manual Aiming Movements in Healthy Young and Healthy Elderly Individuals, and those with Alzheimer's and Parkinson's Diseases.

DEPARTMENT: Department of Psychology, Dalhousie University, Halifax, Nova Scotia

DEGREE: Ph.D. CONVOCATION: October 16 YEAR: 1999

Permission is hereby granted to Dalhousie University to circulate and to have copied for non-commercial purposes, at its discretion, the above title upon the request of individuals or institutions.



Signature of Author

TABLE OF CONTENTS

TABLE OF CONTENTS (Continued)

TABLE OF CONTENTS (Continued)

LIST OF TABLES

LIST OF FIGURES

Continued...

LIST OF FIGURES (Continued)

LIST OF FIGURES (Continued)

LIST OF APPENDICES

LIST OF APPENDICES (Continued)

ABSTRACT

The effect of premovement target location information on the planning and execution of visually directed pointing movements was examined for healthy young and elderly adults, and adults with mild or moderate Alzheimer's disease (AD), or Parkinson's disease (PD). A response cuing paradigm, modified from Posner (1980) was used in two experiments. In both experiments, targets were presented in an arc-like fashion above a visual fixation point at which informational cues (arrow) indicated the probable location of the upcoming target (valid - 60%, invalid - 20%, neutral - 20%). Subjects were instructed to maintain their gaze at the fixation point until the actual target position was indicated and then to point quickly and accurately to the target. In one experiment, the target was indicated by a change at the target position (exogenous orientation) and in the other, was indicated by information presented at fixation (endogenous orientation). Finger position in three dimensional space was sampled by two WATSMART cameras at a rate of 200 Hz and stored on a computer for analyses. Data were analyzed using General Linear Models Repeated Measures Multivariate Analysis of Variance. Group and cue validity were independent variables. Reaction time (RT), movement time (MT), peak velocity (PV), percent deceleration, and resultant error were dependent measures. Overall differences among the groups for RT, MT, and PV were noted. Exogenous overt orienting had a RT advantage for all groups compared with endogenous overt orienting. All groups were able to use cued information to facilitate movements. For exogenous overt movements, healthy elderly, mild AD and PD subjects made all necessary changes to a preplanned program (i.e., invalid cue) prior to movement initiation, with magnitude differences being evident between the groups. The young and the moderate AD subjects made some changes prior to initiation but made adjustments during execution as well, with the AD group taking much longer to initiate and complete movements than the young. For the endogenous paradigm, healthy elderly subjects made all necessary changes prior to initiation while the young and the mild AD groups made some changes prior to initiation and further modifications during execution, with the AD group taking much longer. The PD group made no changes prior to initiation but made all necessary modifications during movement execution. The moderate AD group were not able to perform this endogenous task, suggesting a hierarchy of decline with disease progression. The experiments used were able to distinguish subtle differences in the effects of premovement visual cues on manual aiming movements with the healthy aging process and among AD and PD groups. An exploratory examination of the laterality of target position relative to the body midline and of the meridian effect was conducted.

ACKNOWLEDGEMENTS

I wish to thank my supervisor, John Fisk for his guidance and support during my graduate years. I have appreciated our many insightful and thought-provoking discussions of this work. I also wish to thank my committee members, Ray Klein, John Fentress, and Sherry Stewart for their guidance and feedback throughout this process. I have valued the different perspectives you have provided on this work. On a personal note, I wish to extend my heartfelt gratitude to my family. To my parents, to my husband Bryce, and to our children Danielle and Christopher, my thanks to you for your understanding, and for your love and support throughout these very challenging years.

Chapter 1

GENERAL INTRODUCTION

Responding to external visual stimuli is a critical part of motor performance in our daily lives. Indeed, our survival depends, in part, on our ability to effectively plan, correct, and carry out movements in space (e.g., eating, grasping and manipulating objects). As Posner and Cohen (1980) point out, there is a "beautiful coordination in daily life between hand and eye and attention" (pg. 255). Deficits in visuospatial abilities can have a negative impact on many of our basic activities of daily living (Cummings & Huber, 1992).

Visual attention is clearly linked to motor performance. It has been shown to be important for the detection of an object or an event occurring at a particular spatial location. Posner (1980) postulated that visual orienting must occur before detection is possible, and Navon (1985) pointed out that "to attend is to select for processing" (p. 134). How do attention and information about objects or locations in space affect our movements to those objects or locations? More specifically, how are our hands directed to objects and locations in our environment? We seem "automatically" able to reach out and touch or grasp objects (e.g., light switches, drinking cups, computer keys) with great accuracy, and without any noticeable attention, effort, or cognitive planning. We know that vision specifies the spatial locations of targets and limbs and provides feedback that allows us to make

corrections to movement trajectories (e.g., Abrams, 1992; Carlton, 1992). What other processes or mechanisms influence the manner in which we execute movements? How are these processes or mechanisms affected or influenced by the process of normal aging and by neurodegenerative disorders such as Alzheimer's Disease (considered as primarily a disorder of cognitive abilities) and Parkinson's Disease (considered as primarily a disorder of motor function)?

This thesis investigated the effects of providing advance information and of covert shifts of visual attention on discrete pointing movements (motor programming) with the dominant (right) index finger. Four subject groups were investigated: 1) healthy young individuals, 2) healthy elderly individuals, 3) individuals with Alzheimer's Disease, and 4) individuals with Parkinson's Disease. A paradigm was developed that integrated a covert orienting of visual attention task (modified from Posner, Nissen, & Ogden, 1978) with a manual aiming task. Since manual aiming is an important part of our repertoire of everyday movements, results of this paradigm provide insights into some of the questions about how human movements are produced and how they are influenced by visual attention in the four populations used in this study.

The research on visual attention and motor functioning that has been conducted previously on young healthy individuals will be discussed in the following introductory chapter. The specific literature relating visual attention and

motor functioning with healthy aging, Alzheimer's Disease, and Parkinson's Disease will be discussed in subsequent chapters, each of which is devoted to one specific group. Results of the four groups will be compared and contrasted.

Chapter 2

INTRODUCTION

In order to understand fully the interaction between attention and motor performance, it is necessary to review each of these processes and the current research pertaining to each in some detail.

VISUAL ATTENTION

Attention has been referred to in many contexts. It has been considered to be a selective process, a laborious process, or an alerting and sustaining process (Umilta, 1988). The focus of this thesis will be on visual selective attention to positions in space.

Overt and Covert Shifts of Visual Attention

A good historical overview of research on attention is provided by Wright and Ward (1994). As they describe, the finding that covert shifts of attention are possible was recognized as early as 1866 when Hemholtz noted that he was able to direct his attention to a group of letters prior to having had time to move his eyes to the letter group. In 1890, James pointed out that attention could be controlled either voluntarily (i.e., endogenously) or involuntarily (i.e., exogenously). Following in this vein, Titchner, in 1910, noted that visual attention could be "captured" by the sudden onset of a stimulus (i.e., exogenous stimulus). This historical work provides the basis for current empirical studies on the orienting of visual attention which began in the 1970's, most

notably, the investigations by Posner and his colleagues.

Shifts of visual attention to positions in space can be either overt (i.e., both the fovea of the eyes and attention are directed to a specific area in visual space) or covert (i.e., attention is directed to a specific area in visual space without the eyes moving to foveate a target). Both types of attentional shifts are considered to enhance processing of a change in visual information at that specific position in space (e.g., Posner, Nissen, & Ogden, 1978; Posner, 1980; Posner, Snyder, & Davidson, 1980).

Both overt and covert orienting of attention can be controlled by either exogenous or endogenous means. Exogenous (i.e., involuntary) control is exerted by external sensory stimulation (e.g., a sudden change in the peripheral visual environment), and draws either the individual's attention (i.e., covert orienting) or both the individual's attention and gaze (i.e., overt orienting). In contrast, endogenous (i.e., voluntary) control is exerted by internal cognitive processes. It involves the individual making a voluntary decision to shift attention (i.e., covert orienting) and/or gaze (i.e., overt orienting) to a particular position in space (for a review, see Klein, Kingstone & Pontefract, 1992).

In the current research, endogenous precues (i.e., premovement information presented at a central point in the visual field) designed to elicit covert shifts of visual attention, are paired with target stimuli that elicit overt

orienting (aimed movements) either exogenously (i.e., target location indicated at a peripheral visual location) or endogenously (i.e., target location indicated by information presented at centre).

Posner's Covert Shift of Visual Attention Paradigm

Posner and colleagues (Posner, Nissen & Ogden, 1978) developed a procedure for studying the covert orienting of visually directed attention. In this paradigm, a central visual precue (i.e., endogenous precue) is presented that provides one of three types of information about the location of the future target: a neutral precue (providing no information, indicated by a plus sign), a valid precue (providing correct information, indicated by an arrow), or an invalid precue (providing incorrect information, indicated by an arrow). In order for the informational precue to induce the orienting of attention, the probability that it will provide correct information (valid) must be greater than the probability that it will provide incorrect (invalid) information (e.g., Rafal & Posner, 1987).

The effects of informational precues are examined by conducting "cost-benefit" analyses. These analyses are accomplished by conducting the following comparisons: a) the differences between reaction time (RT) in the valid condition and RT in the neutral condition (termed "benefits"); b) the differences between RT in the invalid condition and RT in the neutral condition (termed "costs"); and c) the differences

between RT in the invalid condition and RT in the valid condition (termed "costs plus benefits") (Posner, Snyder & Davidson, 1980).

"Benefits" refer to the RT advantage provided by the valid precue and are considered to result from the covert shift and engagement of attention to the correct spatial location, prior to target onset (Wright, Burns, Geffen, & Geffen, 1990). This shift and engagement of attention at a specific location in space provides a facilitation (or activation) of that location which allows events to be processed (or detected) more quickly at that location than at non-attended locations (Posner & Snyder, 1975). "Costs" refer to the RT disadvantage on invalid trials and are considered to result from a covert shift of attention to the wrong spatial location and the required disengagement from that wrong location, followed by a shift of attention, and re-engagement of attention at the correct spatial location, prior to responding to the target stimulus (Clark, Geffen & Geffen 1989).

The "cost" and "benefit" analyses are based on the premise that the neutral cue provides only a warning signal about the future target's onset but provides no location information about the future target (Jonides & Mack, 1984; Wright, Richard, & McDonald, 1995). However, it has been questioned whether the neutral cue is "truly neutral" and, therefore, whether it is an appropriate baseline for

calculating separate "costs" and "benefits" (Gawryszewski, Riggio, Rizzolatti, & Umilta, 1987; Jonides & Mack, 1984; Posner, 1986; Wright, Cremona-Meteyard, Geffen & Geffen, 1994). "Costs plus benefits" provide a measure of the time required for the various operations involved in covert orienting of attention. Due to its disregard for the neutral cue, it has been considered by some researchers to be a better measure of the effects of cuing than separate "costs" and "benefits" (Jonides & Mack, 1984; Posner, 1986).

Covert Shifts of Visual Attention

Inherent in Posner's precuing paradigm is the assumption that covert orienting of attention to the correct target location facilitates processing of information at that location (via the activation of neural pathways used to process the future target) and that covert orienting to the incorrect location impedes performance due to the need to disengage, shift and reengage attention on a new target location. This assumption leads to the prediction that RT will be faster in the situation where attention is considered to be correctly oriented (i.e., valid precue condition) compared with the situation where attention is considered to be incorrectly oriented (i.e., invalid precue condition). Consistent with predictions, results of covert orienting of attention studies with healthy young subjects have consistently found RT to be faster to validly precued targets than to invalidly precued targets. In these studies, RT for

neutral precues has often been found to fall between the RT of valid and invalid precues (e.g., Buckolz, Hewey, Khan, & Alain, 1994, experiment 1; Klein & Hansen 1990; Klein & Hansen, 1987, experiments 1 and 2; Posner, 1980; Posner et al., 1980; Shepherd & Muller, 1989, experiment 1; Klein, 1994). However, the finding that RT to neutrally precued targets falls between that of validly and invalidly precued targets has not always held true (e.g., Greenwood, Parasuraman, & Haxby, 1993; Robin & Rizzo, 1992; Tellinghuisen, Zimba, & Robin 1996).

Posner's paradigm and "cost-benefit" analyses have been used to investigate the components of, and the neural substrates involved in, covert orienting of attention.

Cerebral Localization of Covert Orienting Of Visual Attention

Posner, Walker, Friedrich and Rafal (1984) have postulated that the orienting of attention involves three internal processes: a) the disengagement of attention from a location in space; b) the shifting/moving of attention to another location in space, and; c) the engagement of attention on the new spatial location. They consider that the three component processes function in the following manner. When an informational cue is provided, a person will shift attention to that location. If the cue is later determined to be valid, the person will then engage attention to that location. If the cue has been invalid, the person will then be required to

attention to the correct location and then engage it on the actual target. When no information is provided (neutral precue), upon target presentation, the person will shift attention to the target location and then engage attention on the target. This conceptualization allows for the quantitative analysis of these three processes (disengage, shift/move and engage) using the Posner et al. (1978) paradigm and "cost-benefit" analyses. This framework has been used in studies (with both human and non-human animals) investigating the anatomical localization of these three hypothesized component processes involved in covert shifts of visual attention.

The results of numerous studies conducted on non-human animals have linked the parietal lobe with the disengage operation of covert shifts of attention, the midbrain (including the superior colliculus) with the move or shift operation, and the thalamus with the engage operation (for a review, see Colby, 1991). Following these investigations with non-human animals, Posner and colleagues have examined these processes in different groups of humans who have specific lesions to each of these three brain regions.

Posner, Walker, Friedrich and Rafal (1984) conducted experiments that provide support for the notion that the disengage operation is localized to the parietal lobes. Subjects were six individuals with lesions to the right parietal lobe and seven with lesions to the left parietal lobe. Neurological control subjects were three individuals

with frontal lobe lesions (two right and one left hemisphere) and four who had undergone temporal lobectomies for intractable seizures. In their experiments, subjects were presented with a display that consisted of a central fixation box and one box to the left and one box to the right of fixation. Subjects were instructed to maintain their gaze at fixation and to respond to targets (a bright asterisk presented in either the left or the right box) by pressing a key as quickly as possible with the index finger of the hand ipsilateral to the lesioned side. For each trial, an informational precue was presented (the brightening of one of the boxes for 300ms) that was intended to direct the subjects' attention to one of the two peripheral positions. Cues were 80% valid and 20% invalid. The stimulus-onset asynchrony (SOA) was 150ms, 550ms, or 1000ms. Targets remained on until the subject responded or for up to five seconds. Results indicated that, for subjects with parietal lesions, validly cued targets presented on the ipsilesional side had a slight RT advantage over valid targets presented on the contralesional side. RT decreased for valid trials as the SOA increased, and this decrease was equivalent for ipsilesional and contralesional targets. These results led Posner et al. (1984) to conclude that there was no deficit in the shift/move component of orienting of attention for subjects with parietal lesions. The parietal lesion subject group showed a large increase in RT to invalidly cued trials when the cue was ipsilesional and the

target contralesional compared with trials where the cue was contralesional and the target was ipsilesional. Although this difference was noted for both left and right parietal lesion groups, the difference was greater for the right-side lesion group. These differences in the invalid trials were not found in the neurological control subjects. Posner et al. (1984) interpreted this result as an indication of a deficit in the disengage process for individuals with parietal lobe lesions. Posner et al. (1984) suggested that the engage process can be investigated through an examination of the RT differences between the valid targets in the contralesional and ipsilesional fields at long SOA's. When the SOA is prolonged, subjects are considered to have completed the shift/move component and thus have only to engage on the target. Results of their data indicate that, on average, RT to contralesional targets was longer than to ipsilesional targets, for the 1000ms SOA, which would be consistent with a deficit in the engage process. However, Posner et al. (1984) point out that two of the parietal subjects who had disengagement deficits did not have engagement deficits. This led them to conclude that the parietal lobe does not necessarily affect the engage process. The graphic presentation of data by Posner et al. (1984), to illustrate the results of the left and right parietal lesion groups separately, show that the right parietal group had a greater RT difference between the valid trials for contralesional and ipsilesional targets at the

1000ms SOA than at the 150ms and 550ms SOA's. However, the graphs do not include error bars which makes it difficult to comment on the significance of this difference (and these actual values are not reported). The graphic presentation of the left parietal lesion group shows that there were no differences between the valid cues for contralesional and ipsilesional targets at the 1000ms SOA. This would suggest that the deficits in the engage operation are associated more strongly with the right than left parietal lobe. From their results, Posner et al. (1984) concluded that the parietal lobe is "particularly important" for the disengagement of attention and less important for the move and engage operations of attention. An inspection of their data suggests that the right parietal lobe is involved in both the disengage and engage operations and likely in the move operation. However, as the authors point out, results suggest that the greatest influence of the parietal lobes is in the disengage operation.

Posner, Walker, Friedrich and Rafal (1987) conducted a similar study using seven subjects with parietal lobe lesions and ten neurologically intact controls. The experimental set up consisted of a central visual fixation position (a "+" sign) and three boxes on each side of fixation. Cues were a brightening of one of the peripheral boxes. Targets were the filling in of one of the boxes. For invalid trials, the target was always the centre one of the three boxes. Subjects were to respond to the target by pressing a key (with the index finger

of the hand ipsilateral to the lesion) that was positioned at the subject's midline. The parietal subject group showed a RT disadvantage for invalid trials where the target was contra-lesional compared to when the target was ipsilesional. There was no such difference for the control group. This provided further support for the localization of the disengage operation to the parietal lobes.

Rafal, Posner, Friedman, Inhoff and Bernstein (1988) have provided evidence that, they suggest, links the shift/move operation of covert orienting of attention to the midbrain area including the superior colliculus. Subjects were eight individuals with Progressive Supranuclear Palsy (PSP - a degenerative disease that affects subcortical nuclei of the basal ganglia and the brainstem) and eight neurological control subjects with Parkinson's Disease (PD). One feature that distinguishes PSP from PD is that PSP is associated with ophthalmoplegia, particularly for vertical movements. This symptom is associated, in PSP patients, with the degeneration of the superior colliculus and peritectal region. PSP patients have also been shown to have deficits in visually guided behavior (e.g., failure to turn toward others) that is independent of eye movements. Rafal et al. (1988) investigated whether the midbrain areas affected either exogenous or endogenous orienting. They hypothesized that the midbrain was associated with exogenous orienting (which is reflexive and requires no cortical processing) but not with endogenous

orienting (which requires cortical processing). Their paradigm consisted of a central fixation point (a "+" sign) and four boxes (one above, one below, one to the left and one to the right of fixation). Subjects were to press a key with the index finger of the preferred hand when they detected the target (an asterisk in one of the 4 boxes). In the exogenous condition, the precues consisted of a brightening of one of the boxes and were not predictive of future target location (50% probability). This, Rafal et al. (1988) hypothesized, would allow any cuing effects to be attributed to automatic orienting. In the endogenous condition, an arrow precue (pointing to one of the four directions) appeared at fixation. The cue was predictive of future target location (80%) and provided invalid information on 20% of the trials. Any effect of cues in the endogenous condition (since the cues were not associated with a change in luminance and were thought to induce expectancy), were, thus, hypothesized to be due to the endogenous orienting of attention. Orienting in the vertical and horizontal planes were compared. Results indicated that the RT "costs plus benefits" (i.e., invalid minus valid) were smaller for the vertical plane than for the horizontal plane for PSP subjects but not for PD subjects. This difference was due to longer RT in the valid condition in the vertical plane for PSP patients. Rafal et al. (1988) concluded that these results were indicative of a deficit in the shift/move function for the PSP subjects. The effect for the vertical

plane was larger for exogenous than endogenous cues. There was no effect of cue until 150ms SOA for endogenous cues and until 350ms SOA for exogenous cues (to vertical targets). This was compared with an effect of cue at 50ms SOA for the horizontal field for both exogenous and endogenous cues. There was no difference in RT's for invalid trials between the vertical and horizontal planes for the PSP group (indicating no deficits in the disengage operation). Results were interpreted to indicate that midbrain areas are associated with the shift/move operation of covert orienting of attention and have a greater effect on exogenous than endogenous orienting. Rafal et al. (1988) point out, however, that since PSP patients did not orient efficiently to the vertical cues, they may not have performed the disengage operation normally.

Rafal and Posner (1987) found evidence to suggest that the thalamus is involved in the engage operation of attention. As previously stated, the engage operation is investigated by examining the differences in RT to contralesional and ipsilesional targets (for both valid and invalid cues) at long enough intervals between cue and target for attention to have been shifted/moved to the target. Longer RT's to contra-lesional targets compared with ipsilesional targets suggests a deficit in the engagement operation after the shift/move operation is, presumably, completed. Subjects were three individuals with unilateral thalamic damage. The experiment consisted of a central fixation point ("+" sign) with one box

on the left and one on the right of fixation. The cue (valid or invalid) was a brightening of one of the two boxes and the target was an asterisk in one of the two boxes. SOA's were 50ms, 150ms, 500ms and 1000ms. Subjects were to press a key as quickly as possible with one finger of the preferred hand when the target was presented. Results indicated that RT's for contralesional targets were slower than for ipsilesional targets for both valid and invalid trials (i.e., whether attention had been cued to that location or not). This was true even at long SOA's. Rafal and Posner (1987) concluded that the subjects had difficulty engaging attention and that the thalamus is involved in the engagement of attention. Examining the RT for invalid trials compared with valid trials revealed that RT was longer for invalid than for valid trials in the contralesional field for the short SOA's (50ms, 150ms) but not for longer SOA's (500ms, 1000ms). Although longer RT to contralateral invalid targets is consistent with a disengage deficit, this longer RT was not present at longer SOA's, and the difference between the valid and invalid contralateral trials were less than those of the parietal subjects used in other studies (e.g., Posner et al., 1984). This led Rafal and Posner (1987) to conclude that, while the thalamus has a role in the disengagement of attention, this role is less than that of the parietal lobe. They suggested that the thalamus may act indirectly on the parietal lobe to produce a deficit in the disengage operation and that the main

role of the thalamus is in the engage operation.

Results of the work of Posner and colleagues on the localization of the anatomical networks involved in the visual orienting of attention using human subjects with lesions to specific areas of the brain are consistent with results of numerous studies using non-human primates (for a review, see Colby, 1991). The results of the animal and human work in this area led Posner and colleagues (Posner, Petersen, Fox, & Raichle, 1988) to hypothesize that three areas (the posterior parietal cortex, superior colliculus, and thalamus) make up the visuospatial attention system. Further, they propose the following hierarchy of operations (and their related anatomical substrates) in the covert shifting of visual attention: the parietal lobe is responsible for first disengaging attention from its current location; the midbrain then functions to shift/move attention to the target location; and then the thalamus is involved in engaging attention on that new location (Posner and Petersen, 1990, Posner, 1995). While each of these three areas (parietal lobe, midbrain, and thalamus) have been specifically related to the three component operations of covert orienting of attention (disengaging, shift/move, and engage), it is important to recognize that all three areas are intimately interconnected and have numerous afferent and efferent connections with numerous cortical regions (Posner, 1989). This can be seen in each of the human studies reviewed above. Although each of the

three brain areas was considered to be "most" responsible for one of the operations of the covert orienting of attention, the authors pointed out that each was also involved (to a lesser extent) in at least one of the other three component processes.

The results of work on the localization of the cerebral areas associated with the functions involved in the covert orienting of attention have important implications for the investigation of visual attention in groups with known or suspected pathology affecting various subcortical and cortical brain regions, and seems to have merit for developing a better understanding of the behavioral disturbances associated with these disorders. Of particular importance to this thesis is that this research provides the rationale for using subjects with Alzheimer's (AD) and Parkinson's disease (PD). AD is known to involve the parietal lobes (e.g., Haxby, Grady, Horowitz, Schapiro, & Rapoport, 1988) and the thalamus (e.g., Kuljis, 1994). PD is known to have involvement of the midbrain areas (Feldman et al., 1997). The specific pathology associated with Alzheimer's and Parkinson's diseases will be discussed in detail in the relevant subsequent chapters.

MERIDIAN EFFECT

When investigating the allocation of attention in visual space, the topic of the meridian effect is prominent. The meridian effect is an increased "cost" in RT when the cue and target are separated by the vertical or the horizontal

meridian, as compared to when the cue and target are the same distance apart but within a quadrant. The meridian effect assumes that attention has been shifted to the cued location and that a motor response (usually of the eyes) has been prepared. Studies investigating the meridian effect have consistently found this effect to be present in paradigms pairing endogenous precues with exogenous targets (Reuter-Lorenz & Fendrich, 1992; Rizzolatti, Riggio, Dascola, & Umilta, 1987; Umilta, Riggio, Dascola, & Rizzolatti, 1991), and to be absent in paradigms pairing exogenous precues with exogenous targets (Reuter-Lorenz & Fendrich, 1992; Umilta et al., 1991).

Umilta et al. (1991) suggest that the absence of the meridian effect for exogenous (i.e., peripheral) precues is due to an early inhibition process that is triggered by the peripheral precues. This inhibition process has a greater effect when the precue and the target are presented in the same hemifield (i.e., increasing the RT so that it does not differ from situations where precue and target are presented in opposite hemifields). Reuter-Lorenz and Fendrich (1992) have suggested that the lack of a meridian effect for exogenous precues indicates that attentional reorienting and saccadic programming are separate processes that are conducted in parallel. Further, they suggest that attention may be made up of component processes that operate differently with central and peripheral precues in that the spatial

computations needed to make a decision on the location of the precued target may be more complex for central precues than for peripheral precues. This view is similar to that of Klein (1980) and Klein and Pontefract (1992), who view covert orienting of attention and eye movements as separate processes and who have also suggested that exogenous and endogenous orienting behave in distinct manners from each other.

Rizzolatti and colleagues (Rizzolatti, Riggio, Dascola, and Umilta, 1987) suggest that the meridian effect results from changes in motor programs. They propose that, when a cue is presented, a motor program is prepared (usually an eye movement). If the target is then presented at a different location but in the same hemifield as the cue, there must only be a modification in the degree of activation of the previously selected muscle groups. If the target is presented in the hemifield opposite the cued hemifield, however, then different muscle groups than those previously selected must be chosen and activated. Thus, RT to targets in the hemifield opposite the cue are longer due to the additional time required to select and activate new muscle groups compared to making modifications to those already selected (when the cue and target are in different positions in the same hemifield).

To date, the studies of meridian effects have focused on RT paradigms that examine ocular movements and that use a button press for the manual response. As well, the literature to date has been exclusively concerned with manual and

saccadic responses to peripherally presented targets (following either exogenous or endogenous cues). Kinematic paradigms have not yet been used to investigate the meridian effect. This study is the first to use kinematic analyses to investigate the meridian effect and the first to compare responses that do or do not cross the vertical meridian from a cued location (and prepared response) using both peripheral targets and purely endogenous response signals. As this study is the first to examine such issues and the number of trials available to test the meridian effect in this thesis are relatively small, this investigation is considered exploratory in nature.

MOTOR PERFORMANCE

Human motor performance is the result of numerous interactions within a complex system of cognitive processes and physiological mechanisms. In simple terms, this system involves three basic processes. First, information from the environment is received via perceptual sensory mechanisms. Next, this information is processed and a response is planned (via central decision mechanisms). Finally, this planned response is executed via effector mechanisms (Marteniuk, 1976). Feedback (i.e., information an individual receives about their ongoing performance) can play a role in this communication system by allowing an individual to modify a response plan and/or its execution. Whether feedback is used, or how effectively it is used, depends on many factors (e.g.,

type of feedback available, time available to use the feedback, integrity of the systems processing the feedback). When discussing motor response planning and the mechanisms for modifying these plans via feedback, the term most often used is "motor programming". What is meant by this term?

Motor Programming

Like the term "attention", the term "motor programming" has been used in many ways, with no single agreed-upon definition. Motor programming often refers to differing processes depending on the theory or discipline of the investigator (e.g., cognitive psychology, neuroscience, physiology, kinesiology). Shaffer (1992) somewhat aptly states, "the term motor programming is nowadays used so loosely it is in danger of losing its meaning" (p. 181). While the specific actions represented in a motor program are not clear (Semjen & Gottsdanker, 1992), inherent in any definition of the term is the idea that, prior to movement initiation, individuals formulate a movement goal and the details of how the movement will be executed to best meet this goal.

Many theories of motor programming have been proposed. The closed-loop theory postulates that movements are executed by comparing the kinesthetic feedback from an ongoing movement to the "memory" or internal representation of a movement, with errors being corrected when detected. In contrast, the open-loop theory suggests that movements are controlled by central mechanisms without the use of feedback (for a review, see

Summers, 1989). Generally, discrete movements (such as pointing) have been considered to be composed of both an open loop or "ballistic" phase (where the movement is fast with no time for corrections) and a closed loop or error correcting phase (where the movement is slower and corrections can be made based on sensory feedback) (Glencross & Barrett, 1989).

Many models of motor programming suggest that this function is hierarchical in nature. For example, Schmidt (1975) proposed a hierarchically structured generalized motor program theory. This theory is based on a "schema" (Schmidt, 1975) and postulates that the general characteristics of a movement are stored in memory (e.g., joint angles, degree of muscle contraction). According to this theory, a higher level "executive" chooses movement patterns from schema memory and a lower level "effector" executes movements based on these chosen patterns. Arbib (1985) has proposed a distributed control model of motor programming where feedback and feedforward interactions take place at many levels of the central nervous system simultaneously, in what has been described as an "action-perception cycle". Combining these two above-described models has led to the suggestion that the "executive" makes voluntary decisions and detects response selection errors (which take approximately 200 msec to correct) and the lower level "effector" operates in parallel with the "executive" and uses feedback from mechanisms at the spinal-level (for review, see Summers, 1989).

Numerous neurophysiological studies have investigated the anatomical areas associated with differing aspects of motor programming. Upon reviewing and integrating findings from both the information processing and neurophysiological approaches, Requin (1992) outlined a hierarchically organized multilevel system theory of motor programming that relates the information processing stages to neuroanatomical regions. According to his conceptualization, at the highest level (the response selection stage), abstract representations of motor actions are retrieved from memory. This stage of processing has been postulated mainly to involve frontal and parietal association cortex. At the intermediate level (the movement readiness stage), motor actions are specified in terms of general classes of movements rather than in specific terms. This stage has been postulated to involve the premotor cortex, primary motor cortex, and the parietal association cortex. At the lower level (the movement execution stage), motor actions are represented by patterns of activation of motor units. This stage is said to reflect primary motor cortex, premotor cortex, and posterior parietal area. The specific relationship between motor units and peripheral effectors has not been precisely identified. In summary, Requin (1992) suggests that the parietal cortex is involved in response selection, movement readiness, and movement execution while the frontal cortex is involved in response selection only.

Requin's (1992) assertions are consistent with the

results of neurophysiological research such as the studies using single cell recordings in monkeys. For example, both visual dominant and motor dominant neurons have been located in the posterior parietal lobe (Sakata, Taira, Mine, & Murata, 1992) as well as neurons involved in the visual guidance of hand movement (Taira, Georgopoulos, Murata, & Sakata, 1990). Positron Emission Tomography studies of humans performing visually guided pointing movements have also identified enhanced activation of the bilateral parietal cortex during visually guided movements (Grafton, Mazziotta, Woods, & Phelps, 1992). As noted in the previous section, the posterior parietal lobe has also been identified as being involved in covert shifts of visual attention and with the disengagement of attention in particular (e.g., Posner et al., 1984, 1987). Thus, as described by Andersen (1989, 1995) the posterior parietal cortex appears to be neither strictly visual nor strictly motor. Rather, it appears to have the function of integrating visual and motor information (i.e., visual motor processing of information) and also seems to be involved in the orienting of visual attention.

The knowledge provided by motor function researchers regarding the anatomy of motor function has important implications for the investigation of motor function in groups with known or suspected pathology affecting particular brain regions. Combining the knowledge provided by the researchers of visual attention with that provided by researchers of motor

functioning is particularly important when investigating visuomotor integration in clinical groups who have pathology that has been localized to particular brain regions. In this thesis, visuomotor integration (using manual aiming movements) will be examined in two clinical groups who have pathology in brain areas that have been associated with both visual attention deficits and with motor functioning deficits (i.e., AD and PD).

Rosenbaum's Pre-cuing Paradigm

In an attempt to determine the order that individuals program arm movement parameters (i.e., arm, direction, extent), Rosenbaum (1980) developed a "movement precuing" paradigm wherein various amounts of information (i.e., complete, partial, or none) about the requirements of a future movement (i.e., arm - left, right; extent - near, far; direction - forward, backward) are presented prior to a signal to move. This precued information is thought to be used to construct a motor program prior to the instruction to move. Information not included in the precue is thought to have to be integrated into the motor program during the RT interval (i.e., prior to movement initiation). The resulting RT is considered to include the time required to identify the instruction signal to move, to program the values of the movement parameters that were not precued, and to initiate the response (Rosenbaum, 1980).

Results of research using this type of precuing paradigm

have consistently noted shorter RT's to be associated with greater amounts of precued information (e.g., Bock & Arnold, 1992; Bonnet, Requin, Stelmach, 1991; Larish & Frekany, 1985; Rosenbaum, 1980) and thus, have provided support for the proposal that at least some (if not all) motor programming is completed prior to movement initiation. As well, the "direction" parameter was found to take longer to program than the "extent" parameter while the "arm" parameter was found to take the longest amount of time to program (Larish & Frekany, 1985; Rosenbaum, 1980).

Rosenbaum and Kornblum (1982) adapted this precuing paradigm to include valid and invalid precues. This was accomplished by varying which movement parameter (arm, direction, extent) was precued and whether the precue was valid or invalid. For example, direction might be invalidly cued while extent and arm are validly cued. The authors hypothesized that valid cues elicited preprogramming of the response prior to the target presentation and invalid cues elicited reprogramming of the incorrect response at the time the target was presented. Larish and Frekany (1985) found that when "direction" had to be reprogrammed, the complete response was reprogrammed. This, they hypothesized, is because the information about "direction" is required in order for the individual to program the correct pattern of muscular activation of the agonist and antagonist muscles. In contrast, if "extent" has to be reprogrammed, the individual only has to

adjust the force of the muscles that have already been programmed. The authors suggest that this accounts for the longer time required to reprogram responses where "direction" is invalidly cued compared to those where "extent" has been invalidly cued and, thus, has to be reprogrammed.

Although Rosenbaum's (Rosenbaum 1980; Rosenbaum & Kornblum, 1982) precuing paradigms for investigating motor programming are similar in some respects to the precuing paradigm introduced by Posner et al. (1978) for the investigation of the covert shifts of visual attention, studies using these two approaches have been conceptually isolated from each other. For example, while Posner and colleagues refer to "covert shifting of attention", "engagement of attention", "shift of attention", "disengagement of attention" and "exogenous" and "endogenous" sources of information, Rosenbaum and colleagues refer to "advance information", "preprogramming", "programming" and "reprogramming" and do not refer to the precues as having any relationship to attentional shifts.

Nevertheless, both approaches to studies of visually directed movements have used RT (i.e., time from the movement signal to time of movement initiation) as an indication of the time required to plan and execute a motor response and reflect the assumption that such planning is completed prior to movement onset (e.g., Marteniuk, 1976). Thus, variations in RT that result from different movement requirements (e.g.,

distance to be moved, size of target, accuracy requirements) are considered to be indicative of the manner in which motor programming is influenced by changes in the type of response required (Summers, 1989). Despite the importance of the RT interval, however, rich sources of information regarding the way in which information is used in the production of human movement has also come from kinematic analysis of the ongoing movements themselves.

Kinematic Analyses of Visually Directed Movements

Kinematics is the study of movement in space and time. Studies of movement kinematics have generally been conducted by scientists in the fields of physiology, kinesiology, and engineering (e.g. robotics, human factors). These approaches often view motor programming in a slightly different manner from cognitive and information processing approaches. In particular, in the kinematics approach, motor programming is not considered to be completed until the movement has been fully executed. That is to say that any adjustments or changes (i.e., reprogramming) made during the execution of the movement are considered to fall under the domain of motor programming. Thus, the kinematics approach examines the movement trajectory (i.e., path of the movement in space) in an attempt to understand the interaction of sensory and biomechanical factors that affect the execution of a movement.

Kinematic analyses can provide a three-dimensional reconstruction of the movement trajectory and allows for

examination of identified components of movements (e.g., movement duration, peak velocity, acceleration and deceleration times) that closely approximate everyday activities (e.g., reaching for, and grasping, objects). By examining these aspects of the movement trajectory, kinematic analysis has the potential to extend information on motor programming and the manner in which it is affected/influenced by covert shifts in visual attention.

Kinematic studies have provided information about programming during movement execution but not necessarily much about programming prior to initiation because of a lack of integration with other approaches. Kinematic analyses (using discrete movements such as pointing and reaching) have identified three movement phases (i.e., the movement preparation phase, the acceleration phase, and the deceleration phase). During the movement preparation phase (usually defined as the time from the signal to move until actual movement initiation) the necessary motor commands are thought to be assembled. This is followed by the acceleration phase (usually defined as the time from the beginning of a movement until the time peak velocity is reached) where these assembled commands are considered to be initially executed as the limb begins to move toward the target (via the activation of the agonist muscles). During the deceleration phase (usually defined as the time from peak velocity to the time the movement is completed), any necessary corrections or

adjustments are thought to be made as the limb moves to the target (via the activation of the agonist and antagonist muscles) (Abrams, 1992; Brooks, 1986; Glencross & Barrett, 1989). Results of kinematic research have indicated that the duration of each of these phases may vary as a function of the task demands, including the instructions to the subjects. For example, Fisk and Goodale (1989) conducted an experiment where subjects were instructed to move to a target either as "quickly as possible", or as "accurately as possible", compared with a baseline condition (where they were instructed to move both as quickly and as accurately as possible). It was found that movement duration time was significantly longer in the baseline and accurate instruction conditions compared with the fast condition, due to longer deceleration phases for the baseline and accurate conditions. Movement constraints (e.g., target size, target position, and fragility of target) have also been shown to influence the movement trajectory. For example, the deceleration phase has been noted to be significantly longer when pointing to small targets compared with large targets, and when reaching to grasp fragile objects (e.g., round end of light bulbs) compared with sturdy objects (e.g., tennis ball) (Marteniuk, Mackenzie, Jeannerod, Athenes, & Dugas, 1987). The relationship between accuracy demands (e.g., target size) and movement time has been found with such consistency that it has been termed "Fitts Law" (see Sanes & Evarts, 1984 for a review).

Much of the research on changes to movement trajectories has examined the importance of visual feedback. However, kinematic analyses have indicated that changes to movement trajectories can occur by using kinesthetic feedback without simultaneously using visual feedback (Goodale, Pelisson, & Prablanc, 1986; Komilis, Pelisson, & Prablanc, 1993; Martin & Prablanc, 1992; Pelisson, Prablanc, Goodale, & Jeannerod, 1986; Prablanc & Martin, 1992). Support for this notion comes from research that has demonstrated that people are able to make changes to their movement trajectory (for movements that are greater than 400ms in duration) to meet targets that are moved during the time in which they produce a saccadic eye movement to the target. Goodale et al. (1986) had four subjects point to a target that was either displaced or not displaced during a saccade and found that, although subjects did not have a longer movement duration for the displaced targets and exhibited no interruption in the movement trajectory, they were able to reach the target accurately (even though they were unaware of the change in target position). Similar results were found by Pelisson et al. (1986), who suggested that these findings demonstrate the existence of an automatic, continuous, on-line error corrective mechanism for movements, that is able to operate without visual feedback comparisons of limb and target positions. Results of research using variations of this paradigm have led to the assertion that potential terminal

errors are evaluated during the acceleration phase of movements so that there is sufficient time available to make the necessary corrections to the trajectory during the deceleration phase (Komilis et al., 1993).

Such kinematic analyses, which have predominately used healthy young subjects, have provided support for the notion that the programming of more complex, visually directed movements is not completed prior to movement onset by the selection of a series of pre-determined commands. Rather, the movement is conducted, evaluated, and corrected continuously during its execution without the requirement for the individual to be consciously aware of making comparisons between limb and target positions.

While paradigms utilizing kinematic analyses have provided valuable information concerning motor programming and execution, relatively few kinematic studies have attempted to understand the cognitive processes underlying motor programming and execution. Further, while visually directed limb movements have been the predominant type of movement studied, there have been few studies investigating how visual attentional processes affect/influence motor function.

LATERALITY

Many aspects of limb movements are thought to be under the control of the contralateral cerebral hemisphere. For example, the primary motor cortex on each side of the brain controls the movements of the contralateral arm, hand and

fingers, although there is some ipsilateral control of arm movement (e.g., Brinkman & Kuypers, 1973). The contralateral control of finger movements is due to the axons of the finger area of the primary motor cortex projecting to the motor neurons in the spinal cord via the corticospinal tract. As it descends, this tract crosses on each side of the brain stem to the opposite side of the spinal cord (at the midline of the medulla) (Kelly, 1991). Due to this contralateral control of movements, when speaking of the motor programming process, it is difficult to ignore lateralization and hemispheric specialization of function. Laterality of limb function has been examined by comparing individuals' levels of performance on manual tasks for each hand. Those studies using right handed individuals have identified a right hand advantage over the left hand for manual tasks. This right hand advantage for aimed movements in right-handed people has been attributed to the right hand either requiring less corrective movements, or less time to make each correction than the left hand (for a review, see Todor & Smiley, 1985).

Laterality has also been examined by having individuals (usually right handed) reach to a position in space that is either ipsilateral or contralateral to the reaching hand. This research has noted an advantage of ipsilateral over contralateral reaches. Ipsilateral reaches have a faster RT, higher PV, shorter movement duration time, and greater accuracy (e.g., Fisk & Goodale, 1984; Fisk & Goodale, 1985).

Others have noted shorter movement duration, higher PV, and longer percent deceleration for ipsilateral movements, but no differences in RT or accuracy for these movements (Carey, Hargreaves, & Goodale, 1996).

Some researchers have proposed that these contralateral disadvantages may be due to the biomechanical constraints of reaching across the body midline (e.g., more muscle groups are used; larger displacement of the centre of the mass of the limb; higher inertial load) (e.g., Carey et al., 1996). Others have argued that the laterality differences for RT and accuracy are due to the hemifield of target presentation (i. e., due to the need for the interhemispheric transfer of information) while differences in kinematic variables (e.g., PV) are due to biomechanical constraints (Fisk & Goodale, 1985). The RT and accuracy advantage for the ipsilateral hemifield have been suggested to result from the activation of the same hemifield for target identification and for the programming of a response of the hand ipsilateral to the target (i.e., the contralateral left hemisphere). In contrast, contralateral reaches would require the transfer of some information across the hemispheres (e.g., the target presented on the left of midline would be represented by activation of the right primary motor cortex while a movement with the right hand would require the activation of the left primary motor cortex). Thus longer RT for contralateral reaches might be associated with the time required for transfer information

from one hemisphere to the other (Fisk & Goodale, 1985). Motor function studies have not examined the effects of ipsilateral and contralateral reaches in conjunction with covert shifts of visual attention.

VISUAL ATTENTION AND MOTOR PROGRAMMING

As noted previously, despite the somewhat comparable paradigms of Posner et al. (1978) and Rosenbaum (1980) and Rosenbaum and Kornblum (1982), the literature on visual attention and motor programming has remained relatively separate. The reason for this appears to be that early in the study of visual attention (e.g., Posner et al., 1978; Posner & Cohen, 1980) it was recognized that the eye-movement and the hand-movement system were affected differently by the expectancy induced by informational visual precues.

Posner et al. (1978) examined both eye and hand movements (the alignment of a lever with a target) in their investigation of the effects of endogenous visual precues on performance. In experiment 2 of their study, the display consisted of a central fixation cross and a square of light that was presented either to the left or the right of fixation. Endogenous cues were either a plus sign or a left or right pointing arrow. Cues were 80% (valid) and 20% (invalid). The plus sign had no predictive probability. Three conditions were used: 1) move a lever toward the stimulus while maintaining fixation; 2) move the eyes to the stimulus but not the lever; 3) move both the eyes and the lever to the

stimulus. In one experiment the subjects were provided with feedback about their performance in each of the conditions and in a second experiment they were given no feedback. Results indicated that both eye and hand movements showed an effect of cue condition. RT of the hand was, on average, about 100ms longer than RT of eye movements. However, the pattern of performance for the three conditions were similar for both the eyes and the hand. The "costs plus benefits" for the eye movement system were approximately half those of the hand movement system. This was interpreted to be due to the automaticity of the eye movement system. Posner et al. (1978) point out several differences in the effects of expectancy of the eye- and the hand-movement systems. The eye-movement system was more affected by expectancy prior to target presentation (compared with after target presentation) as was evident by errors of anticipation. The hand-movement system had no anticipatory movements. Rather, the hand-movement system was more affected by expectancy after the target was presented (compared with the eye-movement system). This was evident in the large number of directional errors made by the hand system (approximately fives times greater than those made by the eye system). The independence of the eye and hand systems, in this study, was highlighted by the frequency of the eyes and hand moving in different directions. These differences between the eye and hand movement systems, asserted Posner et al. (1978) made it difficult to test

theoretical issues regarding the underlying mechanisms of visual attention.

Posner and Cohen (1980), in a review of work on the eye and hand movement system, point out that while the eye, hand and attention systems had previously been assumed to be separate systems that functioned in a single manner. They, in fact, do not. They are also controlled by different physiological substrates. This author's impression from reading the literature is that, in order to advance theory on the "pure" mechanisms that underlie the orienting of visual attention, there is a need to separate the eye and hand movement systems. Since the eye movement system is closely related to the orienting of visual attention system, further research limited the orienting of visual attention research to eye movements. Often a manual RT measure of target detection is desired to make inferences concerning the effects of informational precues on orienting of attention. It is important to remove the effects of overt response preparations of the hand/limb motor systems from differences in RT as a result of expectancy elicited by visual precues. Thus, a simple key press response was adopted. This response always requires the same motor preparation regardless of cue or target, and thus could not be differentially affected by the cue expectancy.

Similar to the researchers examining visual attention, those researchers investigating motor function appear to have

isolated the preparation of overt responses of the hand/limb from those functions associated with attention. Again, this was presumably in order to advance "pure" theory regarding the physiology underlying motor behavior and its functional responses to specific events unencumbered by other variables (such as attention).

Currently, there continues to remain a noticeable lack of the mention of "shifts of attention" in the cuing paradigms used in motor control research and of "movement programming parameters" (with the possible exception of saccadic eye movement programming) in the visual attention literature. There are, however, some exceptions. For example, Buckolz et al. (1994) presented subjects with informational (i.e., valid, invalid) or neutral precues (and their relative probabilities) in either a 1-response or a 2-response task. The 1-response task was to press a button as quickly as possible (making sure to be accurate) with the right index finger when the target was presented either to the right or left of central fixation. On the 2-response task, subjects were instructed to press a button with the index finger (left or right) that corresponded to the target position (left or right). The authors argued that the 1-response task involved only allocation of attention while the 2-response task involved both attentional allocation and motor programming. Their results indicated that RT for the valid conditions did not differ between the 1-response and 2-response tasks. However, RT for the invalid conditions was

longer for the 2-response than the 1-response task. The authors concluded that this lengthening of RT for the invalid conditions of the 2-response task was due to reprogramming a response and not to attentional allocation. This experiment had the advantage of attempting to integrate the attentional and motor programming research and of challenging the often used procedure of making interpretations and conclusions based on only one perspective. However, the Buckolz et al. (1994) study was limited in that the required motor response was a simple button press (rather than an actual movement of the limb) and that only the RT interval (rather than other variables such as the duration of the movement) was examined. While research of this type is of great value in promoting the integration of the attentional and motor programming perspectives, there remains a gap in the understanding and conceptualization of the interaction of visual attention and the motor programming of more complex limb movements such as reaching movements.

To date, to this author's knowledge, only one experiment has attempted to investigate the effects of visually directed attention to a target on the kinematic parameters of movement in healthy young adults (Elliott & Calvert, 1990). In this experiment, Elliott and Calvert (1990) used a cost-benefit paradigm analogous to that of Posner (1980) and examined RT and total movement duration (MT). They required subjects to move a stylus (as quickly and accurately as possible) to a

target presented either to the left or the right of a central fixation point. Cuing about the possible future position of the target was accomplished via auditory cues (i.e., a high frequency tone cued the right target; a low frequency tone cued the left target). After a two second silence, another tone was presented indicating which target to move to. Results of Elliott and Calvert's (1990) investigation revealed that RT was faster in both the valid and invalid cue conditions than in the neutral condition (although they did not statistically compare valid and invalid cues with neutral cues). The RT for the invalid and valid cues was equivalent. The fact that they found no RT advantage for valid cues compared with invalid cues conflicts with the typical findings cited in the cuing literature (using a simple, entirely programmed motor response - e.g., button press). However, Elliott and Calvert (1990) did find that the MT was longer for movements to invalidly cued targets compared with movements to validly cued targets. This difference in MT implies that, in the invalid condition, a reorganization of the movement took place during movement execution with these "on-line" corrections resulting in an increased time to complete the movement. Thus, "costs" were apparent in MT but not in RT.

While these findings are of considerable interest, Elliott and Calvert's (1990) study had some serious design flaws, which limits its comparability to other studies in which shifts of visual attention have been investigated.

First, there was no direct comparison of the valid and invalid cue conditions with a neutral cue condition. Neutral cues were administered in separate blocks from informational cues, a situation which is not recommended in studies of attentional processes (Jonides & Mack, 1984). The fact that auditory cues (rather than visual cues) were used to direct visual attention also limits the generalizability of the research to the majority of cuing paradigms which have used visual cues. As well, to examine the effect of the availability of visual information on movements, four visual conditions were used: 1) lights on for complete trial (full vision condition); 2) lights extinguished at time of movement initiation; 3) lights extinguished at time of movement instruction; and 4) lights extinguished for 2 seconds prior to movement instruction. These results were collapsed across both tone frequencies and the 4 vision conditions making it difficult for comparisons to be made with results of cuing studies where visual cues have been used to direct visual attention in full vision conditions. Elliott and Calvert's (1990) results however, did provide an indication that a thorough kinematic analysis of visually directed pointing movements might provide greater insight into the effects of shifts of visual attention on the motor programming process.

CURRENT STUDY

The current thesis examined the influence of covert shifts of visual attention on motor programming using a

discrete pointing task. Kinematic analyses were used to elucidate the effects of shifts of visual attention on motor programming. Kinematic measures of the movement trajectory (i.e., movement duration time, peak velocity, percent deceleration, and resultant error) were used in addition to reaction time. Since this study was the first to combine covert shifts of attention, motor functioning, and kinematics, it is considered to be exploratory in nature. While cuing effects on RT (and possibly MT) were expected given the literature on both visual attention and motor functioning, it was unclear whether cuing effects would be evident on any of the kinematic variables when an actual aiming movement to a target was used. General questions included the following. If cuing effects were found for kinematic variables, which kinematic variables would be affected, and what type of cuing effects would be seen in these variables? For example, would MT be longer when provided with incorrect (invalid cue) information (which necessitates a disengagement, shift and reengagement of attention prior to making the movement and/or motor reprogramming) compared with correct (valid cue) information (which allows for the shift and engagement of attention to the correct location and requires no changes in attention or motor programming prior to making the movement)? Would accuracy be greater, less, or equivalent, when attention had been oriented to the incorrect location compared with the correct location? Would the deceleration phase of the movement

be longer than the acceleration phase? If so, would this have any effect on accuracy? These are but a few examples of the types of information that will be examined in the current research. Specific hypotheses for each of the four groups will be stated in a section preceding the method section of each of the four studies. Given that the work is exploratory, and the groups in the study were examined chronologically (i.e., young, elderly controls, AD and PD), the hypotheses for one group may be based on findings from a previous group. The following section will describe the two experimental paradigms used in this thesis.

Experimental Paradigms

The paradigms used in the current study consisted of: 1) endogenous covert shifts of visual attention paired with exogenously generated overt manual movements (using a Posner-type paradigm) - labelled the Exogenous Paradigm; and, 2) the advance provision of endogenous information paired with endogenously generated overt movements (using a Rosenbaum-type paradigm) labelled the Endogenous Paradigm.

The Exogenous Paradigm used in this study is similar to the paradigms used by Posner and colleagues (e.g., Posner et al., 1978) in that a central (endogenous) precue elicits a covert shift of attention to the cued location and the target is presented in an exogenous manner. The Endogenous Paradigm is similar to that used by Rosenbaum and colleagues (e.g., Rosenbaum, 1980; Rosenbaum & Kornblum, 1982) in that the cue

does not elicit a covert shift of attention but rather provides information that can be used in the preprogramming of movements.

The rationale behind such pairings is as follows. The interest of the current research was to investigate the influence of endogenous visual precues (i.e., covert shifts of visual attention) on motor programming. In the Exogenous Paradigm, however, the effects of an endogenous covert shift of attention could not be separated from the effects of response preparation (as the precue provided both information as to where to attend, and what motor response to prepare - given that an actual movement would be carried out). This limitation made it necessary to develop a way to isolate the effects of response preparation from the effects of an endogenous covert shift of attention. The most viable option appeared to be to isolate the effects of response preparation. By eliminating the potential advantage of a covert shift in attention (by presenting all of the target position information at the central fixation point), the Endogenous Paradigm was hypothesized to isolate the effects of response preparation from the potential effects of covert orienting of attention on motor performance. The assumption was that individuals would not shift their attention from fixation when the cue was presented because the information about the future target location would also be presented at this central position. Thus, only the Exogenous Paradigm was expected to

elicit a covert orienting of attention prior to the target location information whereas both paradigms allowed preprogramming of the motor response by indicating the probable future target location. By comparing the results of the two paradigms, it was hoped that the effects of covert shifts of attention on motor performance could be separated from those of response preparation. This would be most likely if covert shifts of attention provided an additive advantage over response preparation only (i.e., if the Exogenous Paradigm provided a greater advantage for the kinematic variables than the endogenous paradigm). Given that it was also possible that there could be differences in the way the exogenously and endogenously presented targets would affect movements, a neutral condition was used in both paradigms. This allowed for an examination of the effects of type of target presentation only (i.e., exogenous or endogenous) as the neutral cues were the same in both paradigms.

Inherent in the rationale for the two paradigms in this thesis is the idea that the preparation of the movement was equivalent in both paradigms. However, it is possible that it may not have been. The following section outlines a number of possible alternative task analyses for each of the cue conditions in each of the two paradigms. In each of the alternatives when motor programming or reprogramming is referred to, it is important to keep in mind that the processes involved may be conducted prior to movement

initiation, both prior to and during movement initiation, or, during movement execution only. These various alternatives may be important when interpreting the results.

Exogenous Paradigm

Alternative #1

Informational Cue Presented: 1) Detect cue; 2) Shift attention to the cued location; 3) Prepare (i.e., "program") a movement to the cued target location.

Target Presented: 1) Detect target; 2) Decision process - was the presented target the same one that was cued and that attention was shifted to and a movement prepared for?

If Yes (i.e., valid cue): 1) Shift gaze to target position; 2) Execute the movement as prepared; 3) Make adjustments as necessary.

If No (i.e., invalid cue): 1) Disengage attention from the cued location, shift it to the target location, reengage attention on the target location; 2) Shift gaze to target position; 3) Reorganize the incorrectly prepared motor program or abort the incorrect program and make a new, correct one; 4) Execute the movement; 5) Make adjustments as necessary.

Alternative #2:

Informational Cue Presented: 1) Detect cue; 2) Shift attention to the cued target location; 3) Do not prepare a movement.

Target Presented: 1) Detect target; 2) Decision process - was the presented target the same as the one that was cued and

that attention was shifted to?

If Yes (i.e., valid cue): 1) Prepare the appropriate movement; 2) Shift gaze to target location; 3) Execute the prepared movement; 4) Make adjustments as needed.

If No (i.e., invalid cue): 1) Disengage attention from the cued location, shift it to the target location, reengage it on the target location; 2) Shift gaze to target position; 3) Program a movement to the target; 4) Execute the movement; 5) Make adjustments as necessary.

Alternative #3:

Informational Cue Presented: 1) Detect cue; 2) Do not shift attention; 3) Prepare a movement to the cued target location.

Target Presented: 1) Detect target; 2) Decision process - was the presented target the same as the one that was cued and that attention was shifted to and a movement prepared for?

If Yes (i.e., valid cue): 1) Shift attention and gaze to target location; 2) Execute the movement as prepared; 3) Make adjustments as necessary.

If No (i.e., invlaid cue): 1) Shift attention and gaze to target location; 2) Reorganize the incorrectly prepared motor program or abort the incorrect program and make a new, correct one; 3) Execute the movement; 4) Make adjustments as necessary.

Alternative #4

Neutral Cue Presented: 1) Detect cue; 2) Divide attention

across all potential target locations; 3) Prepare to move - not to a particular location but merely a readiness to move toward the top half of the computer screen with the right index finger.

Target Presented: 1) Detect target; 2) Disengage attention from all locations, shift attention to the target location, and re-engage it on that location; 3) Shift gaze to the target location; 4) Prepare the remainder of the necessary movement parameters to move to the target location; 5) Execute the movement; 6) Make adjustments as required.

Alternative #5

Neutral Cue Presented: 1) Detect cue; 2) Do not shift attention; 3) Prepare to move - not to a particular location but merely a readiness to move toward the top half of the computer screen with the right index finger.

Target Presented: 1) Detect target; 2) Shift attention and gaze to the target location; 3) Prepare the rest of the necessary movement parameters; 4) Execute the movement to the target location; 5) Make adjustments as required.

Alternative #6:

Neutral Cue Presented: 1) Detect cue; 2) Do not shift attention; 3) Do not prepare a movement.

Target Presented: 1) Detect target; 2) Shift attention and gaze to the target location; 3) Prepare the movement to the target location; 4) Execute the movement to the target location; 5) Make adjustments as necessary.

Alternative #7:

Any Cue Presented: 1) Detect cue; 2) Do not shift attention; 3) Do not prepare a movement.

Target Presented: 1) Detect target; 2) Shift attention and gaze to target position; 3) Prepare the movement; 4) Execute the movement as prepared; 4) Make adjustments as necessary. This alternative is unaffected by the cue validity.

Endogenous Paradigm

Alternative #1:

Informational Cue Presented: 1) Detect cue; 2) Do not shift attention; 3) Program a movement to the target location.

Target Presented: 1) Detect target; 2) Decision process - what is the number that was presented at fixation? Where is the number in the periphery that matches this central number? Is this the target that was cued and that a movement was prepared for?

If Yes (i.e., valid cue): 1) Shift attention and gaze to target position; 2) Execute the prepared movement; 3) Make adjustments as necessary.

If No (i.e., invalid cue): 1) Shift attention and gaze to target position; 2) Reorganize the incorrect motor program or abort the wrong program and prepare a new, correct program; 3) Execute the programmed movement; 4) Make adjustments as necessary.

Alternative #2:

Informational Cue Presented: 1) Detect cue; 2) Shift

attention to the cued location and then shift attention back to the centre of the screen; 3) Gaze remain at fixation; 4) Prepare (i.e., "program") the movement to the cued location.

Target Presented: 1) Detect target; 2) Decision process - what is the number that was presented at fixation? Where is the number in the periphery that matches this central number? Is this the target that was cued and a movement was prepared for?

If Yes (i.e., valid cue): 1) Shift attention and gaze to target position; 2) Execute the movement as programmed; 3) Make adjustments as necessary.

If No (i.e., invalid cue): 1) Shift attention and gaze to target position; 2) Reorganize the incorrect motor program or abort the wrong program and prepare a new, correct program; 3) Execute the movement as programmed; 4) Make adjustments as necessary.

Alternative #3:

Informational Cue Presented: 1) Detect cue; 2) Shift attention to cued location, then shift it back to centre; 3) Do not prepare a movement.

Target Presented: 1) Detect target; 2) Decision process - what is the number that was presented at fixation? Where is this number in the periphery?; 3) Shift attention and gaze to target position; 4) Prepare the movement to the target location; 5) Execute the movement; 6) Make adjustments as necessary.

Alternative #4:

Neutral Cue Presented: 1) Detect cue; 2) Do not shift attention; 3) Become prepared to move (not to a particular location but merely a readiness to move toward the target with the right index finger.

Target Presented: 1) Detect target; 2) Decision process - what is the number that was presented at fixation? Where is this number in the periphery?; 3) Shift attention and gaze to the target location; 4) Prepare the remainder of the movement parameters; 5) Execute the movement; 6) Make adjustments as required.

Alternative #5:

Neutral Cue Presented: 1) Detect cue; 2) Divide attention to all locations, then bring it back to centre; 3) Become prepared to move (not to a particular location but merely a readiness to move toward the target with the right index finger.

Target Presented: 1) Detect target; 2) Decision process - what is the number that was presented at fixation? Where is this number in the periphery?; 3) Shift attention and gaze to the target location; 4) Prepare the remainder of the movement parameters; 5) Execute the movement; 6) Make adjustments as required.

Alternative #6:

Any Cue Presented: 1) Detect cue; 2) Do not shift attention; attention remains at center; 3) Do not prepare a

movement.

Target Presented: 1) Detect target; 2) Decision process - what is the number that was presented at fixation? Where is the number in the periphery that matches this central number?; 3) Shift attention and gaze to target position; 4) Prepare the movement to the target location; 5) Execute the movement; 6) Make adjustments as necessary. This alternative is unaffected by the cue validity.

In summary, this thesis investigated the effects of endogenous informational cues on covert shifts of visual attention and motor programming using a manual aiming movement (with the right index finger) to a visual target. A kinematic analysis was conducted that examined movement duration time, peak velocity, percent deceleration and resultant error in addition to reaction time. The experimental paradigms incorporated informational precues that were intended to elicit either covert shifts of attention in addition to motor programming or motor programming only. Target position information was presented either exogenously or endogenously. The experimental paradigms used in this thesis were intended to bridge the literatures on visual attention and motor programming which have, to date, been conducted in parallel.

An exploratory investigation of the meridian effect on kinematic variables for exogenously and endogenously presented targets was also conducted for the young and for the elderly subject groups. The meridian effect had not yet been

investigated using kinematic analysis, endogenously presented targets, or in elderly subjects. Laterality effects (hemispace of target position relative to the body midline) were also examined. These effects have not previously been examined in conjunction with covert shifts of visual attention.

Since all areas of this thesis are exploratory in nature, it was possible that no effects of cuing would be found for kinematic variables. Therefore, the data for the young subject group was collected and analyzed prior to adding the other groups (healthy elderly, AD,and PD) to the study. The healthy elderly group (compared with the young group) allowed for the examination of the effects of aging on covert shifts of attention and motor programming. As well, the elderly subject group served as a neurologically intact control group for the AD and PD groups. A comparison of the AD and PD groups allowed for the investigation of degenerative diseases that are considered to affect mainly cognitive functions (AD) versus those that are considered to affect mainly motor function (PD). The rationale for the inclusion of the healthy elderly group, and the Alzheimer's and Parkinson's disease groups was based on the combination of the literatures of aging, AD and PD as well as that which has investigated the anatomical regions associated with the orienting of attention and with motor programming. There has been much research conducted that has addressed the effects of aging on motor function and a significant amount of literature exploring the effects of

aging on covert shifts of visual attention. There is only a limited amount of literature for both AD and PD on covert shifts of attention, and, although there are volumes of literature on motor function in PD there is relatively little on AD and motor control. Based on the breakdown of the anatomical correlates of covert orienting of attention and of motor function and on the knowledge of the pathology of both AD and PD, their inclusion in this study is warranted. The rationale for choosing individuals with AD for this study rather than individuals with parietal lobe or frontal lobe lesions (which have often been used in the research examining attention) is that this allowed the comparison of two neurodegenerative diseases. As well, since AD spares the primary motor and primary sensory cortex, inclusion of these subjects does not raise the issues involved in disentangling the effects of the laterality of lesions relative to the laterality of the subject's hand and the target position.

The following chapter provides a description of general methodology and procedures.

CHAPTER 3

METHODS AND PROCEDURES

The methods and procedures used for the four groups included in this study (i.e., healthy young adults, healthy elderly adults, individuals with Alzheimer's Disease and individuals with Parkinson's Disease) were nearly identical. The only exceptions were the number of experimental trials and the number of practice trials per subject.

Prior to beginning the experiment, the procedure was explained to each subject while he/she was simultaneously shown the sequence of events on the computer screen, one step at a time. After it was determined by the experimenter that the subject understood the task, he/she was provided with a number of practice trials. The young healthy subject group were administered 10 practice trials and three blocks of 60 experimental trials each (i.e., 180 experimental trials). The healthy elderly, the AD and PD subject groups were administered two blocks of 60 experimental trials each (i.e., 120 experimental trials). The latter groups were administered fewer experimental trials (than the young subject group) due to concerns for the fatigue of these subjects. Depending on the difficulty that the elderly, AD and PD subjects had in understanding and following the instructions for the experiments, they may have been provided with 20 (and no more than 30) practice trials. No persons were eliminated because of an inability to learn the task in the practice trials

given. Any variations of the basic procedures within each of the four groups are noted in the relevant section of each chapter.

Apparatus and Procedural Techniques

The WATSMART System (Waterloo Spatial Motion Analysis and Recording Technique, Northern Digital, Inc., Waterloo, Ontario, Canada), a three-dimensional (3D) digitizing and motion analysis system, was used. This system operates by tracking the positions of active infrared light-emitting diodes attached to a subject through a pre-calibrated space.

Two high resolution infrared WATSMART cameras were positioned 3.0 meters apart, 1.8 meters from the floor, and at a perpendicular distance of 2.4 meters from the 21 inch computer monitor on which the experimental trials were administered. During the experiments, the positions of two infrared light-emitting diodes (IRED's) (one affixed to the subject's right index finger and one affixed to the subject's right index knuckle) were monitored by the two WATSMART cameras. Two-dimensional coordinates were collected on-line by each of the two cameras. Three-dimensional IRED positions were then reconstructed off-line by the WATSMART data collection system and were stored using a COMPAQ 386s/20 computer for analysis.

The WATSMART system was interfaced with another 386 personal computer which controlled the sequencing of the cue and target information that was projected to the computer

monitor on which the experimental stimuli were presented.

Calibration

Camera Position

To calibrate the positions of the WATSMART cameras, a 53 x 53 x 72 cm rigid metal calibration frame in which 20 IREDs are embedded was provided with the WATSMART system. This calibration frame was positioned in the centre of the work space (i.e., the space where the computer monitor would be positioned). As described by Haggard and Wing (1990, p. 317) "Because the relative positions of these IREDs are known, the cameras' different views of the frame are used to calculate the cameras' locations relative to the frame, using a direct linear transformation method. This calculation specifies a calibration map for each camera independently. Mapping permits subsequent conversion of raw data, comprising the location of each IRED as it appears in two dimensions to each of the two cameras, into three dimensional coordinates".

The WATSMART software calculates a calibration error which is the average error between the true dimensions of the frame and those determined by the least squares fitting procedure that yields the cameras' positions (Haggard & Wing, 1990, p 317). This error is expressed as a root mean square (RMS) measure. The average RMS calibration error in this study was 0.62mm (*SD* = 0.08mm). This is consistent with other studies with this methodology in which the RMS is generally found to be less than 2mm (Carey, Hargreaves, & Goodale, 1996;

Elliott, Carson, Goodman & Chua, 1991; Jakobson & Goodale, 1991).

Target Position

Four target positions were used in each experimental paradigm. The positions of each of the four targets (presented on the computer monitor screen during the experiment) relative to the cameras was determined for each subject by having the subject point to the centre of each individual target as accurately as possible during a "calibration" condition of no time constraints. The positions of the IRED that was affixed to the tip of the subject's right index finger was recorded for two seconds and the averaged three-dimensional position was used to define the "calibrated" target position. This allowed the subjects to determine where they perceived the centre of the target to be located and accounted for variations in IRED placement across subjects. Later in the analysis, resultant error (i.e., 3-dimensional) was calculated (by WATSMART software) for each trial in reference to these calibrated target positions.

Data Collection

All trials were initiated by the experimenter's keypress command and required that the subject have his/her finger pressing down on a computer mouse switch which served as the start position. With the experimenter's keypress, the sequencing computer generated the previously determined randomized cue/target sequence and produced an external

trigger signal to initiate the collection of data by the WATSMART system.

The subject's mouse switch was interfaced with the sequencing computer via a single channel of an A-D converter (WATSCOPE, Northern Digital Inc.) which was used to detect premature anticipatory movements (i.e., finger movements prior to the signal to move) by the subject. Such anticipatory movements resulted in cancellation of the trial as described in the "Experimental Procedure" section (p. 67).

During each trial, the positions of the finger IREDs were recorded at 5ms intervals (i.e., 200 Hz) for four seconds by the WATSMART system beginning with the experimenter's keypress.

Data Reduction

Data were reduced following the experimental sessions. For each trial, the two sets of raw two-dimensional linearized camera data were converted to three-dimensional cartesian coordinates by direct linear transformation and filtered at 15 Hz using a second-order dual-pass Butterworth filter to remove high-frequency noise. Resultant error, a composite error measure reflecting deviations from the target in three dimensions was calculated for the final finger IRED position at the defined end of movement (i.e., finger IRED velocity <5 cm/sec for 10 successive frames).

The filtered three-dimensional displacement data (for each of the two IRED's) was differentiated to provide a

velocity profile. Reaction time, peak velocity, time from movement onset to time of peak velocity, movement end time, and resultant error, were derived from these velocity profiles. Instantaneous velocity was calculated on the basis of distance travelled over three successive frames and was expressed in cm/sec. Each velocity profile (for each of the two IRED's) was examined visually to identify any position errors resulting from IRED reflections or errors in the calculation of movement onset due to changes in hand position that preceded moving the finger toward the target. IRED #1 (i.e., finger position) was examined to determine end of movement and IRED #2 (knuckle position) was examined for all other dependent variables. This was necessary because, for some individuals, IRED #1 was occasionally obscured from the WATSMART cameras during some portions of the movement. The end point of the movement, could be accurately calculated from IRED #1 since finger velocity dropped to zero and remained at this value when the subject contacted the computer screen with his/her finger. IRED #2 (the knuckle IRED) was not obscured from the cameras during the movement and data from this IRED were used to calculate all other dependent measures. Pilot data also suggested that IRED #2 typically moved prior to IRED #1 and therefore provided a better indication of "true" movement onset. Any trials where recording errors were noted were manually corrected (if the trajectory profile was smooth enough to make this possible) by the investigator by

substituting values according to the criteria that were set for each of the dependent variables, outlined in the following section. Trials that could not be corrected (due to extensive reflection errors resulting in extensive noise in the trajectory) by this procedure were discarded. The percentage of trials that were discarded in any analysis did not exceed 2.3%.

Dependent Measures

The time of end of movement and resultant error were derived from the finger position data collected from IRED #1. All other measures were derived from the data collected from IRED #2, which was placed on the knuckle of the subject's index finger.

Reaction time was defined as the time from target onset to the time of movement initiation and was recorded from IRED #2. The criteria for this reaction time measure was the time at which the movement velocity reached 5cm/sec and remained so for 10 successive frames (i.e., 50ms).

Movement duration time was defined as the time from movement initiation until the time that the final finger position was recorded (i.e., finger IRED velocity < 5cm/sec for 10 successive frames). Movement duration was calculated by subtracting the time at the end of movement from IRED #1 from the time of the movement onset from IRED #2. Movement accuracy was defined as the resultant error (absolute error in mm) between final finger position (IRED #1) relative to target

position as defined by finger position on the target calibration trials. A normalized measure of deceleration time was provided by the time from peak velocity to the time of the end of the movement, expressed as a percentage of the total duration of the movement.

Eye Movement Monitoring

Eye movements were monitored by the experimenter via a Panasonic video camera (model # WV-BP310) with a Cosmicar Electronic Control Zoom Lens that provided a focus on the subjects' eyes. This camera was mounted on a tripod, positioned 66cm to the left of the computer monitor and 130cm from the floor. A Panasonic black and white video monitor (model # TR-930CB) was positioned on the desk in front of the experimenter to allow a constant view of the subject's eye position. Subjects were required to maintain fixation from cue onset to target onset. Since cue onset was initiated by the examiner and target onset could be determined by a luminance change (i.e., reflection from the subject's face), the examiner could ensure that the subjects did not change their eye position from the central fixation point until target position was indicated. Any trials in which subjects' changed their eye position prior to the target indication were discarded and were repeated.

Experimental Procedure

Experimental Set-Up

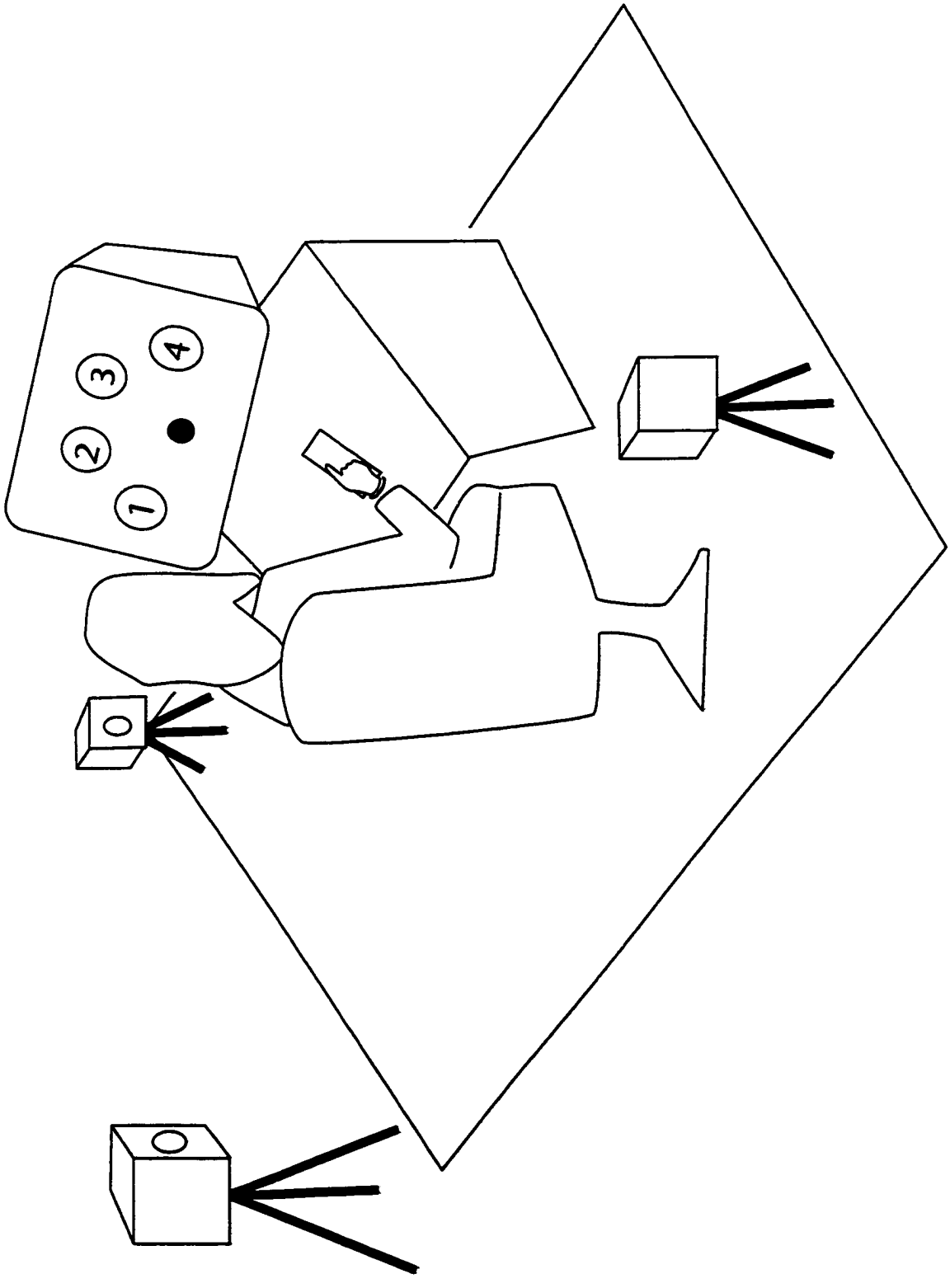
Work Space Set-up

WATSMART cameras are designed to detect all types of infrared light and recognize each IRED as the centre of all the infrared light entering the camera lens. Thus, the cameras cannot distinguish between the infrared light coming from an IRED and a reflected source of infrared light (e.g., natural sunlight, incandescent light). As a result, the two-dimensional IRED coordinates that are recorded are based on the light arriving at the cameras via reflected paths as well as light coming directly from the IRED. In order to obtain accurate measurements of the position of the IREDs, it was imperative to reduce irrelevant infrared light.

In order to reduce errors due to reflections from the IREDs, the walls surrounding the computer monitor were painted with a flat black paint, black blinds covered the windows, and the table on which the computer was positioned on as well as the chair used in the experiment were covered with a black material. The face of the computer monitor was covered with a translucent black foam. Subjects wore a black hospital-type gown over their clothing. Low room lighting was provided by a halogen floor lamp positioned to the right of the video monitor. All the above measures were taken to reduce any possible reflections during testing and reduce visual distraction in the environment (see Figure 3.1).

Figure 3.1

Figure 3.1 illustrates the experimental set-up used in this thesis. The two WATSMART cameras are pictured behind the subject. The Panasonic video camera that provided a focus on the subjects' eyes (and monitored eye position) is illustrated to the left of the subject and the computer monitor on which the experiments were administered is illustrated in front of the subject. The computer systems and the video monitor used by the experimenter were positioned behind the subject and are not depicted in this illustration.


4
3
2
1

Experimental Trials Set-up

On the upper half of the 21 inch computer screen, four possible targets (consisting of the numbers, 1, 2, 3, and 4) were displayed in an arc-like fashion above a central fixation point. All potential targets were equidistant from the central fixation point (16cm). The positions of the four targets were chosen so that the distance reached by the hand would be equivalent to all positions. The numbers (for target positions and for central cues) were 7mm high and 6mm wide at their widest point. The circle surrounding the numbers was 10mm in diameter. The arrow that provided the informational cue was 15mm long with the arrow-head 5mm in length on each side. The vertical line of the "plus sign" (neutral cue) was 15mm and the horizontal line was 10mm. Targets were 10cm apart. While head position was not fixed, subjects' heads were positioned approximately 50cm from the screen. At the central fixation point, informational cues were presented that were later determined to be either valid (providing correct directional information with respect to subsequent target position, designated by an arrow and occurring in 60% of the trials) or invalid (providing incorrect directional information, designated by arrow and occurring in 20% of the trials). Uninformative (neutral) cues (designated by a "+") occurred on 20% of the trials.

Target position and cue type were counterbalanced within each trial block. Following 10 practice trials, three blocks

of 60 experimental trials each were presented (for the young subject group). Each block consisted of 12 neutral trials (3 in each of the 4 positions), 36 valid trials (9 at each of the 4 positions), and 12 invalid trials (3 at each of the 4 positions). Table 3.1 provides a breakdown of the number of combinations of cue and target positions by hemispace.

Table 3.1

Number of Trials of each Cue/Target Combination Across 3 Experimental Trial Blocks Combined

	POSITION CUED				
	1	2	3	4	"+"
TARGET POSITION					
1	27	3	3	3	9
2	3	27	3	3	9
3	3	3	27	3	9
4	3	3	3	27	9

Note: The diagonal (where cue and target are identical) represents valid trials. The last column (cue = "+") represents neutral trials. All other locations indicate invalid trials.

Experimental Procedure

The procedure used in the two experimental paradigms (i.e., Exogenous and Endogenous) was similar. Subjects were seated in the darkened room facing the 21 inch computer monitor on which the experiments were administered. Subjects had one IRED (IRED #1) affixed (with clear adhesive tape) close to the end of their right index finger and another (IRED #2) to the knuckle of their right index finger.

On each trial, subjects were instructed to depress the start switch, to look at, and to maintain their gaze at the central fixation point until the actual target position was indicated. Subjects were instructed to reach out and touch the target with their right index finger quickly and accurately as soon as it was indicated. Trials in which the subject moved his/her eyes from the fixation point prior to target onset (as determined by the experimenter) were repeated. Trials in which subjects exhibited anticipatory finger movements (i.e., finger released start switch prior to cue being extinguished) were detected by the computer and aborted by the program. In the case of anticipatory movements, an auditory "beep" sounded and the message "Moved Too Soon" appeared on the screen. The trial was then repeated as the next subsequent trial.

In both the Exogenous and Endogenous Target Paradigms, the cue appeared at the fixation point and remained for 750ms. In the Exogenous Target Paradigm, the target was a circle appearing around one of the peripheral digits (see Figure

3.2). In the Endogenous Target Paradigm, circles appeared around all the digits from the beginning of each trial, and the target was the appearance of a digit at fixation (see Figure 3.3). This procedure ensured that the target, at which the pointing movement was directed in the two paradigms was physically identical (digit with a circle).

Figure 3.2

Figure 3.2 illustrates the exogenous target paradigm. Valid, neutral and invalid conditions are shown. The subject was instructed to maintain his/her gaze at the centre fixation point. One of the three types of cue was presented at that point and remained on for 750ms. The cue then extinguished and was followed immediately by a circle surrounding the actual peripheral target. At that time, the subject was to reach out and touch the target with their right index finger quickly and accurately.

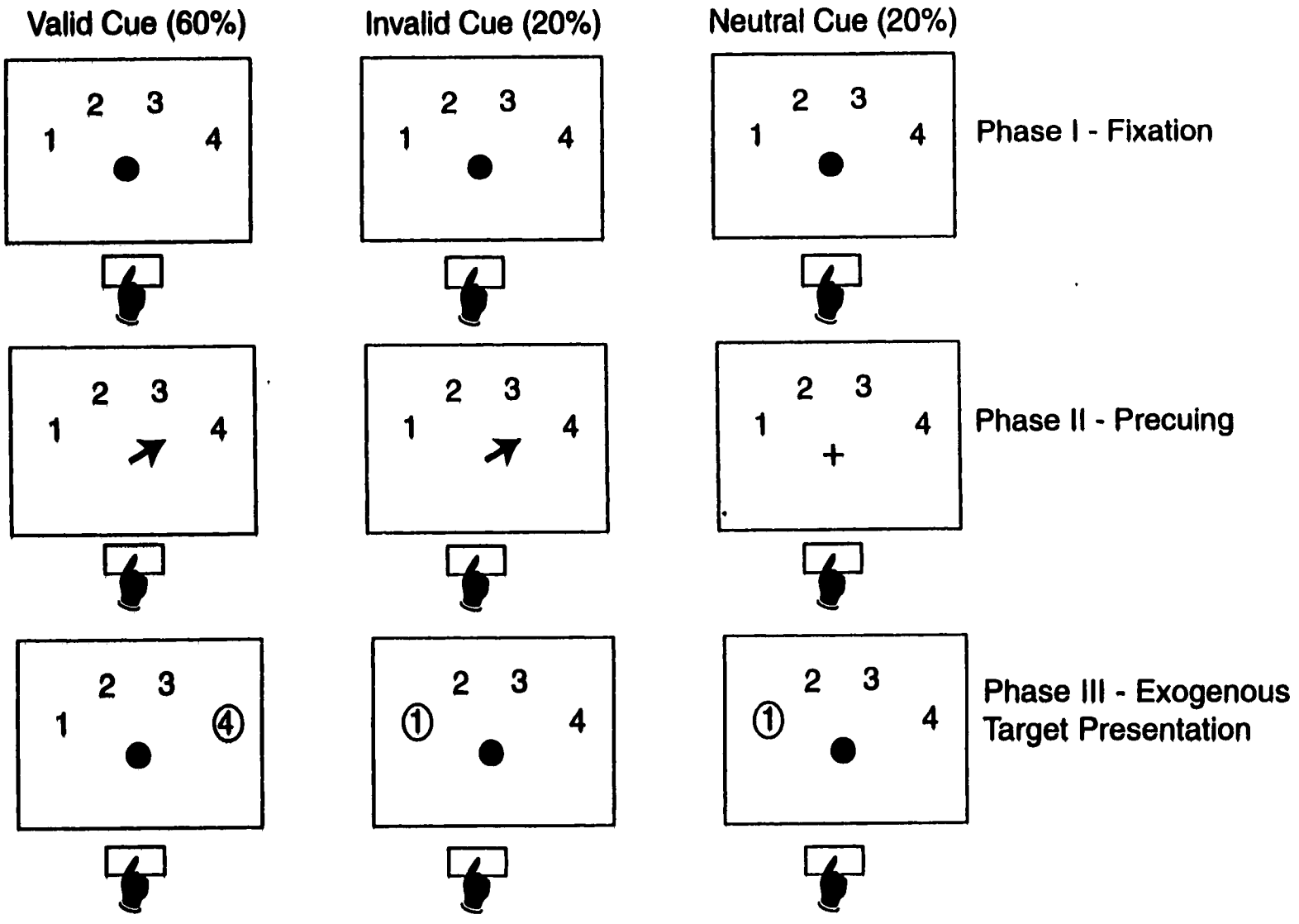

Valid Cue (60%)
Invalid Cue (20%)
Neutral Cue (20%)
2 3
1 4
Phase I - Fixation
Phase II - Precuing
+
Phase III - Exogenous
Target Presentation
①
④

Figure 3.2

Figure 3.3

Figure 3.3 illustrates the endogenous target paradigm. Valid, neutral and invalid conditions are shown. The subject was instructed to maintain his/her gaze at the centre fixation point. One of the three types of cue was presented at that point and remained on for 750ms. It then extinguished and was followed immediately by a number presented at the central fixation point that indicated the actual peripheral target. At that time, the subject was to reach out and touch the target with their right index finger quickly and accurately.

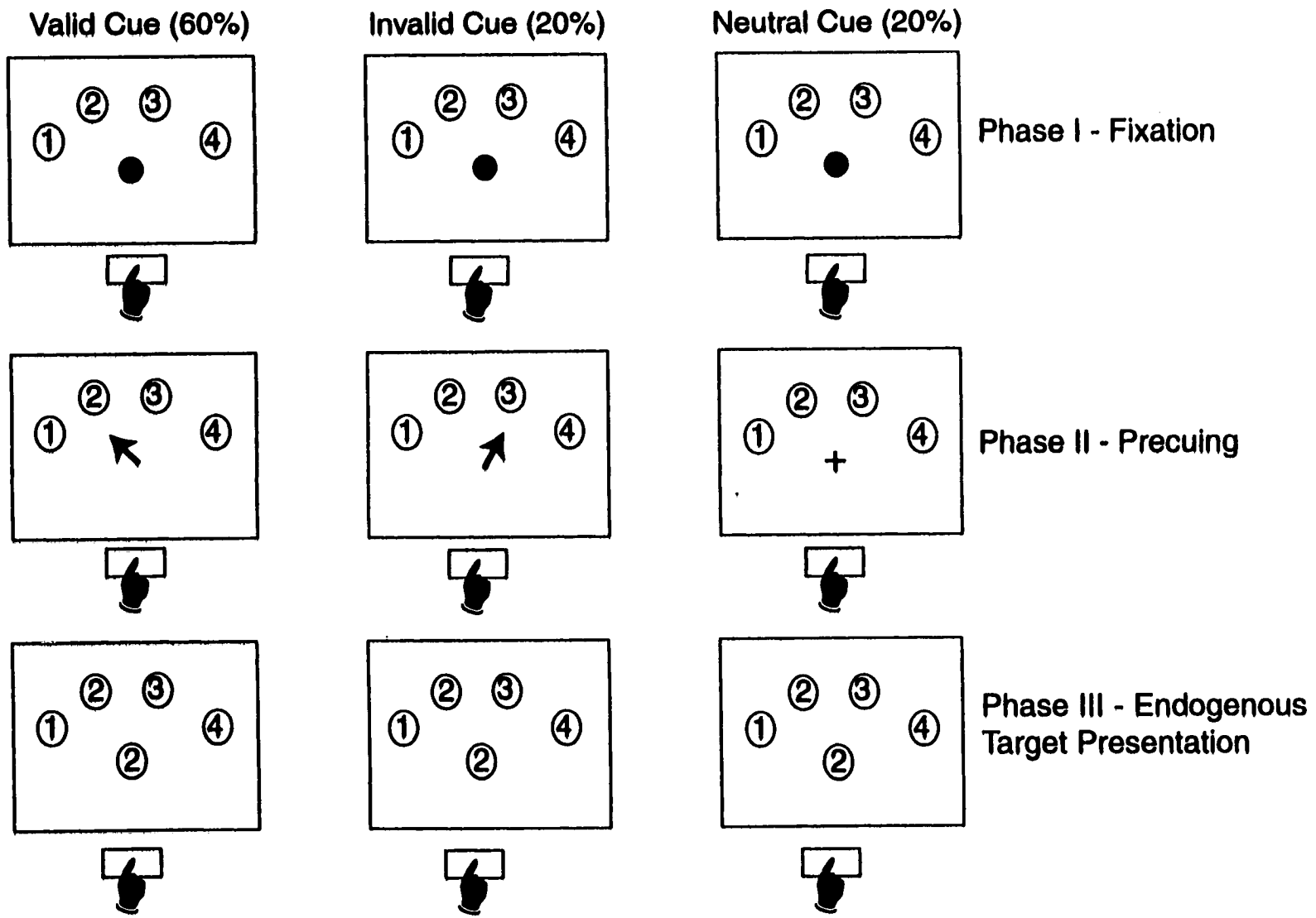

Valid Cue (60%)
Invalid Cue (20%)
Neutral Cue (20%)
Phase I - Fixation
Phase II - Precuing
Phase III - Endogenous Target Presentation
1
2
3
4

Figure 3.3

STATISTICAL ANALYSES

Measures for each dependent variable were calculated on a trial by trial basis, for each subject and for each experimental condition. Data were analyzed using General Linear Models Repeated Measures Analysis of Variance (MANOVA) procedure (SAS Institute Incorporated, Cary, North Carolina). Pillai's trace was used as the measure of multivariate significance as it was considered to be a robust criterion given the nature of the data (unequal number of observations for the dependent measures) (Tabachnick & Fidell, 1989).

The General Linear Model is a form of multiple regression that is used when there are several independent and dependent measures, some of which may be correlated with each other. It uses least squares adjusted means. These means are calculated when there is unequal data among the cells in the analysis and are adjusted based on an estimate of what the means would be if the cells had an equal number of observations. This estimate is calculated using the least squared error solution: the estimated values are those that would result in a regression line that has the smallest total squared errors. The standard error of estimate of the least squares adjusted means is used as the measure of variability. It is a measure of the standard distance between the regression line and the actual data points and provides an indication of how accurately the regression line predicts the observed values. MANOVA only uses trials for which there is data for all of the

dependent measures for a particular trial. Any trials that had data for one dependent measure missing were discarded. That is to say that, in the present analysis, only trials that had data for RT, MT, PV, time from peak velocity to end of movement, and resultant error were included in the MANOVA analysis.

For situations where there were no a priori planned comparisons, significant multivariate effects and interactions were followed up with univariate ANOVA's (using the least squares means data from the MANOVA). Where univariate ANOVA's were significant, post-hoc comparisons were conducted using t-tests on the least squares adjusted means. In situations where hypotheses had been generated based on the literature (i.e., a priori comparisons had been planned), the univariate ANOVA's and/or t-tests were examined even in the absence of significant multivariate effects. In all tests of significance a standard alpha level of 0.05 was adopted. Univariate outliers were identified based on the averaged error being 4.5 SD or greater from the least squares mean error. These trials were checked for equipment (i.e., reflection) errors or data entry errors and necessary corrections made. No outliers were removed (other than those resulting from equipment errors), as such trials were considered to reflect natural variance in movement.

The studies presented in this thesis were conducted in the chronological order that is presented. The study for the

young subject group was completed first, followed by the elderly subject group, the AD subject groups and the PD subject group. Each of the subject groups were first analyzed individually. The elderly subject group was then compared with the young subject group, the AD group was compared with the elderly group, and the PD group was compared with both the elderly and the AD groups. The elderly group was used as a control group for both the AD and PD groups. Examining differences between the young and elderly subject groups allowed the investigation of the effects of aging on visual attention and motor function. Examining differences between the AD and PD groups allowed the investigation of changes in motor function and attention based on cognitive versus motor involvement of diseases that affect the elderly.

Chapter 4 - Study 1

THE EFFECTS OF VISUAL ATTENTION AND MOTOR PROGRAMMING ON MANUAL AIMING MOVEMENTS IN HEALTHY YOUNG ADULTS

METHOD

Subjects

Subjects were 15 healthy young adults (8 females, 7 males) who were students at Dalhousie University, or staff and volunteers of the Queen Elizabeth II Health Sciences Centre. All were right-handed as indicated by self-report and by a score of at least 5 out of 7 on Kimura's Handedness Questionnaire (Kimura, 1986). All reported having normal or corrected to normal vision. Mean age was 28.6 (SD = 4.4) with a range of 24 to 39 years. Mean level of education was 17.6 (SD = 1.8) with a range of 13 to 20 years. Informed consent was obtained from all subjects prior to testing in accordance with the procedures approved by the Queen Elizabeth II Health Sciences Centre Research Ethics Committee.

Procedure

Refer to Chapter 3 for details of the procedure.

Analyses

Statistical analyses were conducted as described in Chapter 3. Three separate analyses were conducted on the data for the healthy young subjects. Analysis 1 examined the effect of cue validity (i.e., valid, invalid, neutral) and target type (i.e., exogenous - circle surrounded peripheral target; endogenous - number that corresponded to target presented at

centre) on the dependent variables. Analysis 2 examined the effect of laterality (i.e., target presented in the left vs the right hemispace) on the dependent variables. Analysis 3 examined the meridian effect (i.e., cue and target presented in the same vs the opposite hemispace) on the dependent variables. Details of each analysis will be provided in the subsequent sections.

HYPOTHESES

Cue Condition

For the young subject group, a number of hypotheses regarding cuing effects were developed based on the literature of visual attention, motor programming and kinematic analyses. They were as follows:

Cues that provide valid information were hypothesized to result in a RT and MT advantage (compared with cues that provide invalid information) for movements of both the Exogenous and Endogenous Paradigms. RT (and possibly MT) for neutral cue conditions were expected to fall between the valid and invalid conditions. If the hypotheses for faster RT and shorter MT for valid compared with invalid cue conditions held true, then it was possible that the PV would have been higher in the valid compared with the invalid conditions. Again, if MT was longer for invalid vs valid cue conditions, it was possible that the deceleration phase would have been longer for the invalid cue condition compared with the valid condition (as the changes required in the invalid condition to

allow an accurate movement were likely to have been made during this phase). If the deceleration phase was longer for invalid than valid cue conditions, then it was expected that the accuracy (i.e., resultant error) would have been equivalent in the two conditions. If the deceleration phase was equivalent in the two cue conditions, it was likely that the resultant error would have been greater in the invalid condition due to insufficient changes being made in the final stages of the movement to allow the finger to intercept the target accurately.

RT was expected to be faster, overall, for the Exogenous Paradigm compared with the Endogenous Paradigm. This hypothesis was based on the RT research that has found RT to be faster to exogenous than endogenous cues (see Klein & Briand, 1986 for a review).

"Costs plus benefits" was expected to be larger for the Exogenous compared with the Endogenous Paradigm. Larger "benefits" were expected because there would have been no need to shift attention or program a movement when the target was indicated in the valid condition of the Exogenous Paradigm. In contrast, there would still have been a need to shift attention to the target position prior to having made the movement in the Endogenous Paradigm (thus, less benefit of the cue). There would also have been larger "costs" for the Exogenous over the Endogenous Paradigm because (in invalid conditions) there would have been the requirement to

disengage, shift and reengage attention plus make changes to the motor program when the target was presented while this would not have been required in the Endogenous Paradigm.

Laterality Of Target Position Effect

RT was expected to be faster, MT shorter, PV higher, percent deceleration shorter, and resultant error smaller, for movements to targets presented in the ipsilateral hemispace (i.e., right) than to the contralateral hemispace (i.e., left). Percent deceleration was expected to be shorter to targets presented in the ipsilateral compared with the contralateral hemispace.

Meridian Effect

There was expected to be a meridian effect for the Exogenous Paradigm but not for the Endogenous Paradigm. This is based on the notion that a shift of attention is necessary for the meridian effect, and it was hypothesized that there would have been no shift of attention in the Endogenous Paradigm of the current study. However, given the exploratory nature of this analysis, the Endogenous Paradigm was also examined for a meridian effect.

The meridian effect was expected to be found for RT for the Exogenous Paradigm (i.e., RT was expected to be faster for trials where the cue and targets were on the same side of the vertical meridian compared to those where the cue and target were on opposite sides of the vertical meridian). Since no kinematic variables had ever been examined for the meridian

effect, no specific hypotheses could be made for these variables (i.e., MT, PV, % deceleration, resultant error). However, given the exploratory nature of this analysis, all variables will be examined for meridian effects.

ANALYSIS 1

CUE CONDITION

Analysis 1 was a 3 (Cue Condition - Valid, Invalid, Neutral) x 2 (Target type - Exogenous, Endogenous) Multivariate Within Subject Repeated Measures Analysis of Variance Design (MANOVA) using a SAS General Linear Models procedure. Dependent measures were reaction time (RT), movement duration time (MT), peak velocity (PV), percent deceleration, and resultant error.

"Costs plus benefits" (i.e., invalid minus valid cue condition) was examined using planned paired comparisons. The least squares adjusted means were examined to determine the differences in the "costs plus benefits" for the Exogenous and for Endogenous Paradigms for each of the dependent variables (i.e., RT, MT, PV, percent deceleration, resultant error).

Results

The multivariate MANOVA revealed a multivariate effect of cue condition, $F(10, 50) = 5.30$, $p < .0001$, and of target type, $F(5, 10) = 8.53$, $p < .005$. There was a marginal multivariate cue condition by target type interaction $F(10, 50)$, $= 1.99$, $p = .05$. The significance of the multivariate effects allowed the inspection of the univariate repeated measures Analysis of Variance (ANOVA) for each of the five dependent variables.

Reaction Time (See Figure 4.1)

Results of the univariate repeated measures ANOVA on RT indicated that there was a main effect of cue condition, $F(2, 28) = 35.24$, $p < .0001$. Post-hoc comparison t-tests using the least squares adjusted means (collapsing across target type) indicated that the mean RT for the valid condition (M = 386.9ms, SE = 5.9ms) was significantly faster than that of the neutral condition (M = 477.1ms, SE = 10.3ms; $t = 7.56$, $p < .0001$) and the invalid condition (M = 451.7ms, SE = 10.3ms; $t = 5.42$, $p < .0001$). RT for the neutral and the invalid cue conditions did not differ significantly from each other, although there was a trend for the neutral condition to have longer RT than the invalid condition ($t = 1.74$, $p = .09$).

There was also a main effect of target type, $F(1, 14) = 52.13$, $p < .0001$, with the Exogenous target type having a RT advantage which averaged 75.9ms over the Endogenous target type (M for Exogenous target type = 400.6ms, SE = 7.4ms; M for Endogenous target type = 476.5ms, SE = 7.4ms; $t = 7.22$, $p < .0001$). A cue condition by target type interaction, $F(2, 28) = 5.15$, $p < .01$ was evident. Although the pattern of results were the same for the two target types (i.e., Valid < Invalid < Neutral), there was a difference in the magnitude of the "costs plus benefits" between the target types. The "costs plus benefits" for the Exogenous Paradigm was 55.3ms ($t = 7.94$, $p < .0001$) and was 74.3ms for the Endogenous Paradigm ($t = 10.65$, $p < .0001$). A statistical contrast examining the

difference between the "costs plus benefits" for the two target paradigms was marginally significant (F = 3.69, p = .06). The effects of target type did not differ in each of the cue conditions. The RT for the valid conditions was faster for the Exogenous compared with the Endogenous Paradigm (t = 12.12, p < .0001). The RT for the neutral conditions was faster for the Exogenous compared with the Endogenous Paradigm (t = 10.43, p < .0001). The RT for the invalid condition of the Exogenous Target Paradigm was faster than the invalid condition of the Endogenous Paradigm (t = 9.22, p < .0001).

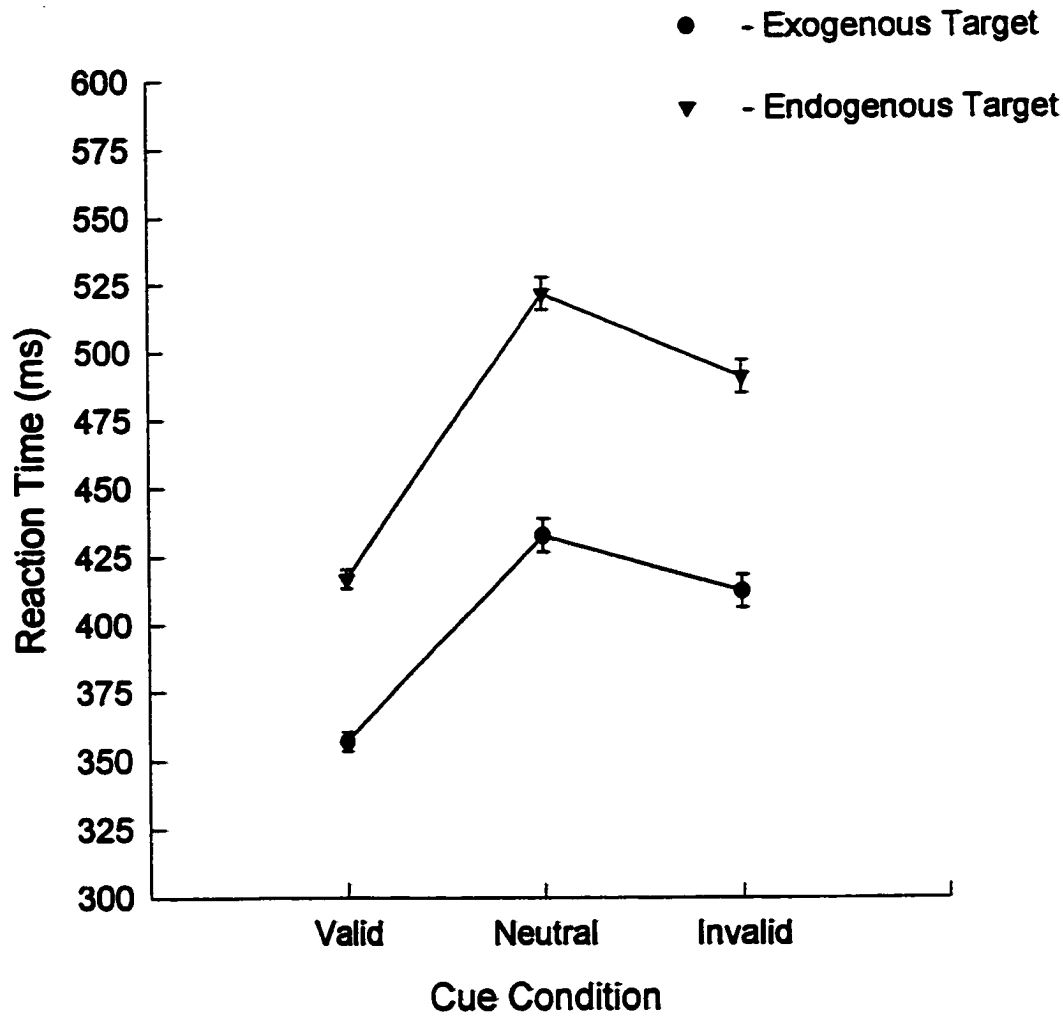


Figure 4.1 - Reaction time as a function of cue condition and target paradigm. The error bars represent the standard error of the least squares means.

Movement Duration Time (See Figure 4.2)

Results of the repeated measures ANOVA for MT revealed a main effect of cue condition, $F(2, 28) = 7.92$, $p < .002$. Post-hoc comparison t-tests using the least squares adjusted means (collapsing across target type) indicated that the mean MT for the valid condition (M = 576.3ms, SE = 2.7ms) was significantly less than that of the invalid condition (M = 597.3ms, SE = 4.7ms; $t = 3.82$, $p < .0007$). Mean MT's differed only marginally between the valid and the neutral (M = 587.3ms, SE = 4.7ms) conditions ($t = 2.01$, $p = .05$) and were equivalent for the neutral and invalid conditions ($t = 1.48$, $p = .15$).

There was no main effect of target type on MT, $F(1, 14)$, $= .19$, $p = .67$ (M for Exogenous target type = 583.9ms, SE = 9.8ms; M for Endogenous target type = 590.0ms, SE = 9.8ms, $t = 0.44$, $p = .67$) but there was a significant cue condition by target type interaction, $F(2, 28) = 4.26$, $p < .05$. An examination of the cue conditions within each of the two target types (using the least squares adjusted means) revealed different patterns of results for the two target types. In the Exogenous Paradigm, the valid cue condition had a shorter MT (M = 577.8ms, SE = 2.2ms) than the invalid condition (M = 590.1ms, SE = 3.8ms; $t = 2.76$, $p < .01$) although neither differed from the neutral condition (M = 583.8ms, SE = 3.8ms; both p values > .05). In the Endogenous Paradigm, however, the valid cue condition had a shorter MT (M = 574.8ms, SE = 2.2ms)

than both the invalid (*M* = 604.5ms, *SE* = 3.8ms; *t* = 5.99, *p* < .0001) and the neutral conditions (*M* = 590.8ms, *SE* = 3.8ms; *t* = 3.61, *p* < .0001). The neutral condition also had a shorter MT than the invalid condition (*t* = 2.50, *p* < .02). Thus, the magnitude of the "costs plus benefits" for MT was less in the Exogenous (12.3ms) Paradigm than in the Endogenous (29.7ms) Paradigm (*F* = 7.65, *p* < .01). Also contributing to the interaction was the difference in target paradigm for each of the cue conditions. For the valid and neutral conditions, the effect of target type was not significant (*t* = 0.95, *p* = .35 for the valid conditions; *t* = 1.28, *p* = .21 for the neutral conditions). For the invalid conditions, MT was shorter for the Exogenous (*M* = 590.1ms) compared with the Endogenous (*M* = 604.5ms) Paradigm (*t* = 2.64, *p* < .01).

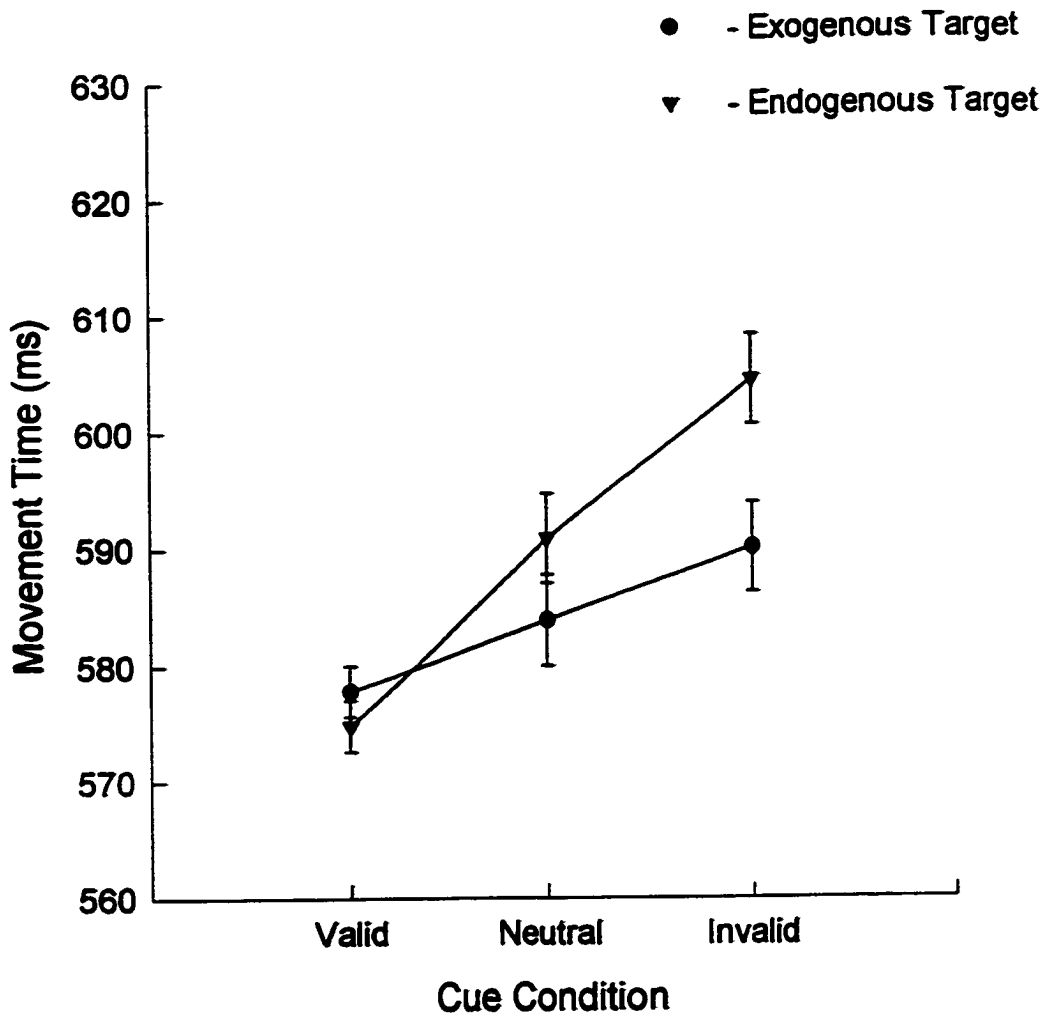


Figure 4.2 - Movement time as a function of cue condition and target paradigm. The error bars represent the standard error of the least squares means.

Peak Velocity (See Figure 4.3)

Results of the univariate repeated measures ANOVA revealed a main effect of cue condition, $F(2, 28) = 11.68$, $p < .0005$. Least squares adjusted means (collapsing across target type) indicated that the valid condition had a slightly higher peak velocity (M = 91.1cm/sec, SE = 0.2cm/sec) than both the invalid (M = 89.0cm/sec, SE = 0.3cm/sec; $t = 4.71$, $p < .0001$) and the neutral conditions (M = 90.1cm/sec, SE = 0.3cm/sec; $t = 2.22$, $p < .05$) with the neutral condition having a marginally higher PV than the invalid condition ($t = 2.03$, $p = .05$). There was no main effect of target type, F (1, 14), = .00, $p = .98$ (M for Exogenous target type = 90.1cm/sec, SE = 1.3cm/sec; M for Endogenous target type = 90.0cm/sec, SE = 1.3cm/sec; $t = 1.34$, $p = .20$).

There was a marginal cue condition by target type interaction, $F(2, 28) = 3.08$, $p = .06$. Given this marginal interaction, cue condition within each of the target types was examined with post-hoc comparisons (t-tests) using the least squares adjusted means. These revealed slightly different patterns of results for the two target types. For the Exogenous Paradigm, the valid condition had a higher peak velocity (M = 90.6cm/sec, SE = 0.3cm/sec) than the invalid condition (M = 89.3cm/sec, SE = 0.5cm/sec; $t = 2.37$, $p < .05$) while neither differed from the neutral condition (M = 90.2, SE = 0.4cm/sec; both p values > .05). In the Endogenous Paradigm, the valid condition had higher PV (M = 91.6cm/sec,

SE = 0.2cm/sec) than both the invalid (*M* = 88.6cm/sec, *SE* = 0.4cm/sec; *t* = 5.51, *p* < .0001) and the neutral conditions (*M* = 89.9cm/sec, *SE* = 0.4cm/sec; *t* = 3.00, *p* < .006). The invalid and the neutral conditions differed only marginally, *t* = 2.05, *p* = .05. Thus, while the "costs plus benefits" for PV were only 1.3cm/sec (*p* < .05) for the Exogenous Paradigm, they were 3.0cm/sec (*p* < .0001) for the Endogenous Paradigm. These differences in the "costs plus benefits" between the two target paradigms was statistically significant (*F* = 4.95, *p* < .05).

Also contributing to the interaction was the difference in target paradigm for each of the cue conditions. For the invalid and neutral conditions, the effect of target type was not significant (*t* = 1.15, *p* = .26 for the invalid conditions; *t* = 0.45, *p* = .66 for the neutral conditions). For the valid conditions, PV was higher for the Endogenous (91.6cm/sec) compared with the Exogenous (90.6 cm/sec) Paradigm (*t* = 2.45, *p* < .02).

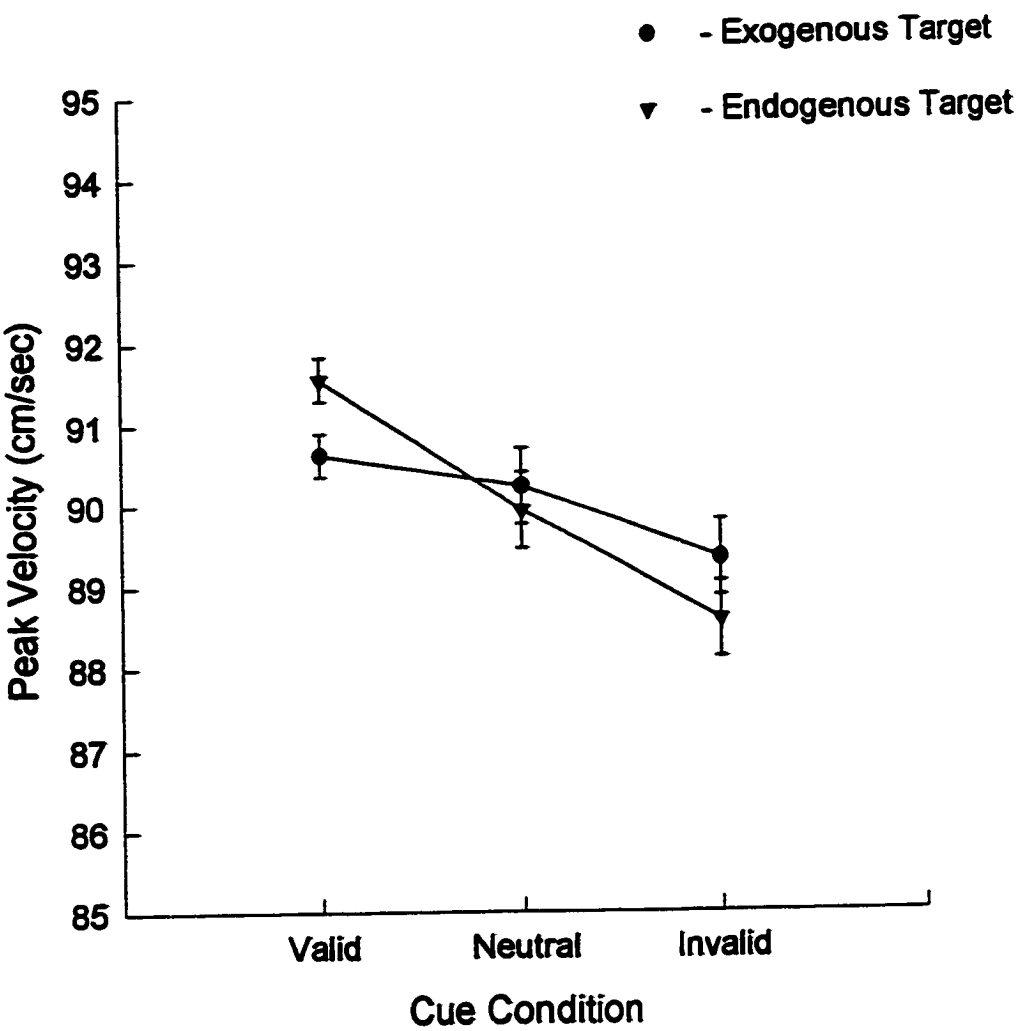


Figure 4.3 - Peak velocity as a function of cue condition and target paradigm. The error bars represent the standard error of the least squares means.

Percent Deceleration (See Figure 4.4)

Results of the repeated measures ANOVA for percent deceleration indicated that there was a marginal main effect of cue condition $F(2, 28) = 3.29$, $p = .05$. Post-hoc comparisons indicated that this marginal main effect was accounted for by a significant difference between the valid ($M = 61.1\%$, $SE = 0.2\%$) and the neutral ($M = 61.9\%$, $SE = 0.3\%$) conditions ($t = 2.40$, $p < .02$) (collapsing across target type) with a higher percentage of the movement completed in the deceleration phase in the neutral compared with the valid condition. No other significant differences among cue conditions were evident.

There was no main effect of target type, $F(1, 14), = 1.79$, $p = .20$ (M for Exogenous target type = 61.1%, $SE = 0.5\%$; M for Endogenous target type = 62.0%, $SE = 0.5$cm/sec). No cue condition by target type interaction, $F(2, 28) = 2.03$, $p = .15$ was evident. Planned comparisons using the least squares adjusted means indicated that there were no significant "costs plus benefits" for the Exogenous target type (0.13%, $t = 0.41$, $p = 0.68$). The "costs plus benefits" for the Endogenous target type was statistically significant ($t = 2.71$, $p < .01$) but was small in magnitude (0.9%). The differences in the "costs plus benefits" between the two target paradigms was not significant ($F = 2.66$, $p = .11$).

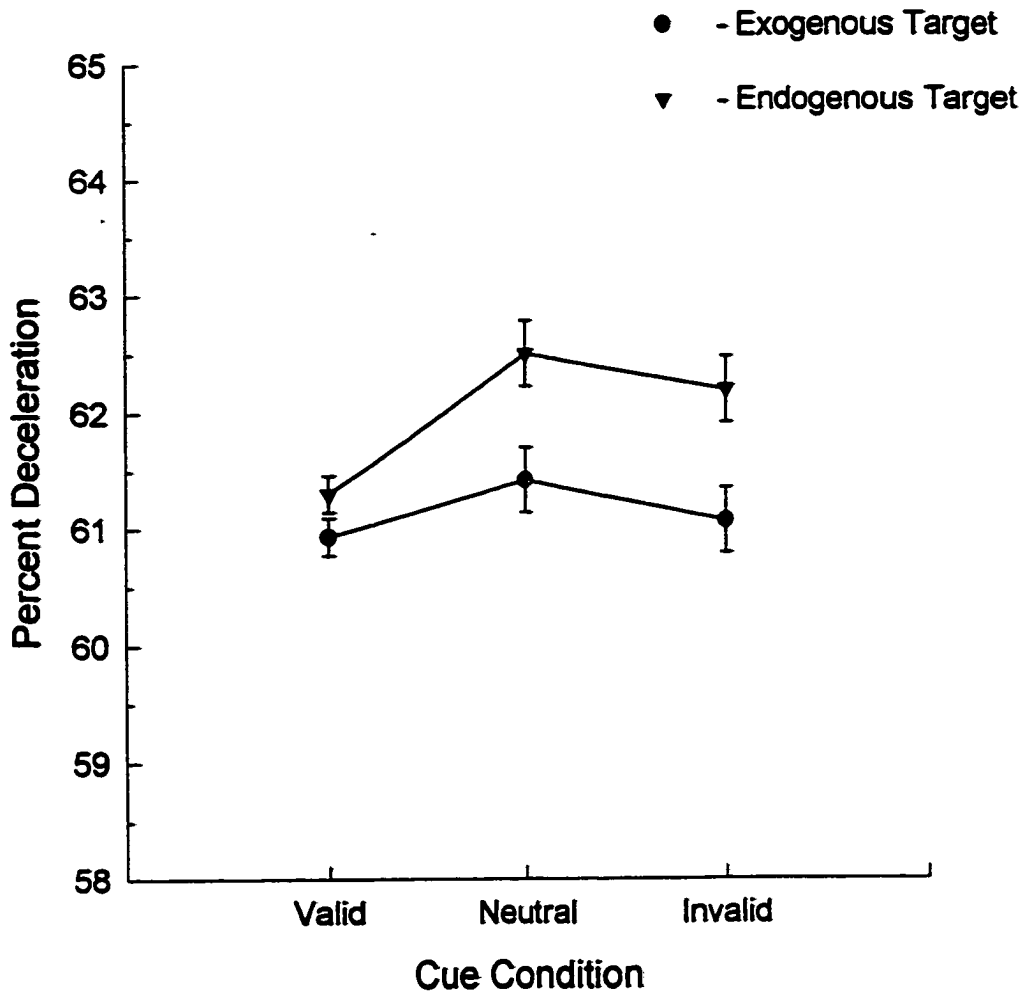


Figure 4.4 - Percent deceleration as a function of cue condition and target paradigm. The error bars represent the standard error of the least squares means.

Resultant Error (See Figure 4.5)

Results of the repeated measures ANOVA indicated there was no main effect of cue condition, $F(2, 28) = .17$ $p = .85$, no main effect of target type, $F(1, 14) = 2.91$, $p = .11$, and no cue condition by target type interaction $F(2, 28) = .80$, $p = .46$. There were no "costs plus benefits" for either the Exogenous Paradigm ($t = 0.29$, $p = .77$) or the Endogenous Paradigm ($t = 1.21$, $p = .24$). Least squares adjusted means resultant error ranged from 7.8mm to 8.0mm for the Exogenous Paradigm and 7.4mm to 7.7mm for the Endogenous Paradigm.

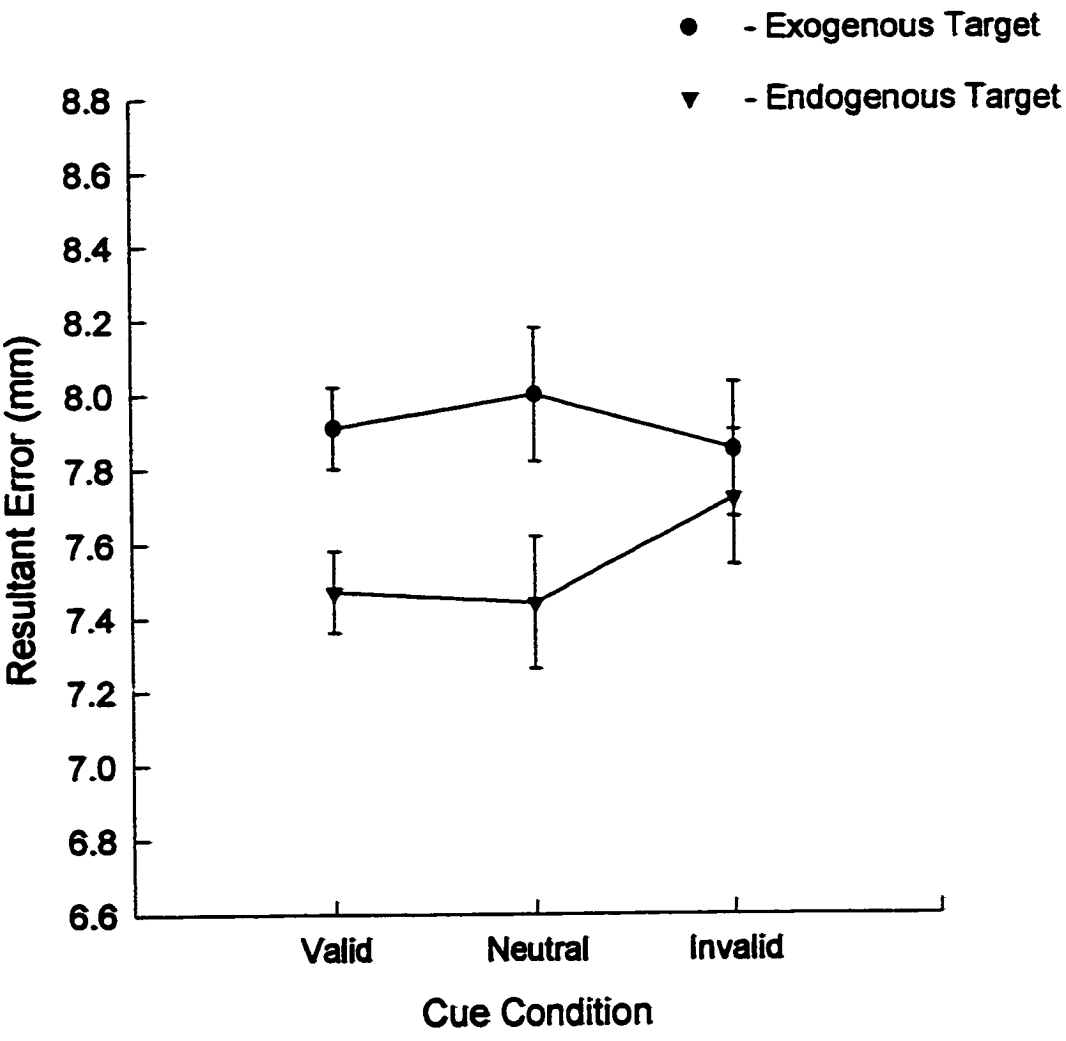


Figure 4.5 - Resultant error as a function of cue condition and target paradigm. The error bars represent the standard error of the least squares means.

Neutral Cues

The RT advantage for Exogenous targets in the current study are best illustrated by direct comparison of the neutral cue conditions for the two target paradigms. This comparison revealed that RT was approximately 89ms faster (t = 10.43, p < .0001) for the Exogenous compared with the Endogenous Paradigm in the absence of any confounding effects of covert shifts of attention or advance motor programming information.

The results of the neutral cue conditions for the dependent variables often failed to fall between those of the valid and invalid conditions. Thus, the value of the neutral cues, in the current study, appeared to be their ability to provide the means for a direct comparison of the Exogenous and the Endogenous Paradigms, rather than as a basis for calculating "costs" and "benefits" within the target conditions. Comparing the remaining dependent variables on the neutral cue conditions of the two target paradigms (using the least squares adjusted means) indicated that percent deceleration was slightly shorter (t = 2.72, p < .01) and resultant error was slightly greater (t = 2.17, p < .05) for the Exogenous Paradigm compared with the Endogenous Paradigm. However, there was no significant difference in MT or PV. Thus, exogenously presented targets had a RT advantage and a slight disadvantage in accuracy compared with endogenously presented targets. The fact that RT was slower for the Endogenous Paradigm but MT and PV did not differ between the

two paradigms suggests that the differences in information processing requirements for the two types of targets occurred prior to the initiation of the movement. The most likely difference in the two paradigms appeared to be in the additional decision process in the Endogenous Paradigm (i.e., interpreting the numeric digit presented at the centre to indicate the target position and translating this into a movement to the corresponding peripheral target). This finding has important implications for the interpretation of the results of the two paradigms.

ANALYSIS 2

HEMISPACE OF TARGET POSITION

The analysis used to examine the effects of hemispace of target position relative to the body midline (i.e., left vs right) was a one-way Multivariate Repeated Measures Analysis of Variance. Dependent measures were RT, MT, PV, percent deceleration, and resultant error. A series of a priori planned paired comparisons using the least squares adjusted means was conducted to examine the differences between the hemispace variables.

Two variables were created for this analysis. "Target Left" included all trials for which the targets were either "1" or "2". "Target Right" included all trials for which the targets were either "3" or "4". A preliminary analysis indicated that there were no interactions among target type and hemispace of target position; therefore, the hemispace variables were collapsed across both experimental paradigms (Exogenous and Endogenous) to provide greater statistical power for the analysis. All cue types were collapsed for this analysis as counterbalancing would negate any differences as a function of cue as there were the same number of valid, invalid and neutral cues on each of the left and right sides.

Results (See Tables 4.1 and 4.2)

The multivariate MANOVA revealed a multivariate effect of hemispace of target position, $F(5, 10) = 22.89$, $p < .0001$. The significance of the multivariate analysis allowed the

inspection of the univariate mixed model Analysis of Variance (ANOVA) for each of the five dependent variables.

Results of the univariate repeated measures ANOVA's revealed significant main effects of hemispace of target position for RT, $F(1, 14) = 7.49$, $p < .05$, for MT, $F(1, 14) = 73.82$, $p < .0001$, for PV, $F(1, 14) = 50.26$, $p < .0001$, and for percent deceleration, $F(1, 14) = 8.81$, $p < .01$. There was a marginal main effect of hemispace of target position for resultant error, $F(1, 14) = 4.38$, $p = .06$.

In contrast to most studies, which have found RT to targets presented in the hemispace ipsilateral to the moving hand to be faster than that to targets in the hemispace contralateral to the moving hand, examination of the least squared adjusted means indicated that movements to targets in the left hemispace (i.e, contralateral) were initiated significantly faster than those to targets in the right hemispace (M for target left = 414.8ms, SE = 1.7ms; M for target right = 421.2ms, SE = 1.7ms; $p < .05$). Since this result is at odds with those of previous research (e.g., Fisk & Goodale, 1984, 1985), a measure of RT derived from the WATSCOPE data file was also examined (i.e., mouse release by the finger as opposed to kinematically derived measure of RT [i.e., knuckle IRED movement]). However, the results of this data analysis indicated no differences in RT as a function of hemispace of target presentation [$F(1, 14) = 0.62$ $p = .62$; M for target left = 450.5ms, SE = 1.6ms; M for target right =

449.3ms, SE = 1.6ms].

Movement duration was significantly shorter for movements to targets in the ipsilateral compared with contralateral hemispace (M for target left = 606.4ms, SE = 3.9ms; M for target right = 559.1ms, SE = 3.9ms, p < .0001). PV was significantly higher to targets in the ipsilateral compared with the contralateral hemispace (M for target left = 85.3cm/sec, SE = 1.0cm/sec; M for target right = 95.6cm/sec, SE = 1.0cm/sec). Percent deceleration was significantly longer to targets in the ipsilateral compared with the contralateral hemispace (M for target left = 59.8%, SE = 0.8%; M for target right = 63.0%, SE = 0.8%). Resultant error tended to be slightly greater to targets in the ipsilateral compared with contralateral hemispace (M for target left =7.3mm, SE = 0.3mm; M for target right = 8.1mm, SE = 0.3mm; p = .06).

The possibility that collapsing the cue conditions had some influence on these results was considered (although counterbalancing makes this seem unlikely). However, an analysis conducted using reaches of neutral cues only provided the same pattern of results: once again, the overall multivariate main effect of hemispace of target position was significant, F(4, 11) = 15.70, p < 0002. As illustrated in Table 4.2, the pattern of results for the neutral cues was similar to that for all cues combined.

Table 4.1

Hemispace of Target Position Effect (all cue conditions)

	RT (Ired 2)	RT (WATSCOPE)	MT	PV	%Decel	Error
Target Left	414.8[a]	450.5	606.4[b]	85.3[b]	59.8[a]	7.3[c]
SE	1.7	1.7	3.9	1.0	0.8	0.3
Target Right	421.2[a]	449.3	559.2[b]	95.6[b]	63.0[a]	8.1[c]
SE	1.7	1.7	3.9	1.0	0.8	0.3

Same letter suprascripts indicate significant differences.

[a], $p < .05$

[b], $p < .0001$

[c], $p = .06$

Table 4.2

Hemispace of Target Position Effect (neutral cues only)

	RT (Ired 2)	RT (WATSCOPE)	MT	PV	%Decel	Error
Target Left	470.2[a]	506.7	610.5[b]	84.8[b]	60.3[a]	7.1[c]
SE	3.7	3.4	4.9	1.0	0.8	0.3
Target Right	483.9[a]	514.6	563.9[b]	95.2[b]	63.5[a]	8.1[c]
SE	3.7	3.4	4.9	1.0	0.8	0.3

Same letter suprascripts indicate significant differences.

[a], $p < .05$

[b], $p < .0001$

[c], $p = .05$

ANALYSIS 3

MERIDIAN EFFECT

Since the hemispace of target position effects have been noted for the pointing movements used in this study, there was the need to examine the effect of meridian without confounding hemispace of target position issues. To control for this, two sets of comparisons were conducted. One comparison examined the meridian effect for contralateral reaches (i.e., target left of centre), and one examined the meridian effect for ipsilateral reaches (i.e., target right of centre). Each of these two comparisons included a contrast of the situation in which the cue and target were in the same hemispace (i.e., within-hemispace) with the situation in which the cue and the target were in the opposite hemispaces (i.e., between-hemispace).

Since the cuing literature is characterized by a gradient of performance around the cued location (e.g., Downing & Pinker, 1985; McCormick & Klein, 1990), it is necessary to assess the meridian effect using targets that are equated for distance between the cue and target. Therefore, to investigate the meridian effect for contralateral targets, a within-hemispace cue and target condition (cue = 1, target = 2) was compared with a with a between-hemispace cue and target condition (cue = 3, target = 2). To investigate the meridian effect for ipsilateral targets, a within-hemispace cue and target condition (cue = 4, target = 3) was compared with a

between-hemispace cue and target condition (cue = 2, target = 3).

Since the meridian effect has previously been noted only for endogenous cues paired with exogenous targets (e.g., Reuter-Lorenz & Fendrich, 1992; Rizzolatti et al., 1987; Umilta et al., 1991), it was deemed appropriate to compare each of the two target paradigms individually. Thus there was a within- and between-hemispace comparison for ipsilateral and for contralateral targets for both the Exogenous and the Endogenous Paradigms (i.e., 4 separate analyses).

Each analysis was a one-way Multivariate Repeated Measures Analysis of Variance (MANOVA). Dependent measures were RT, MT, PV, percent deceleration, and resultant error. A series of a priori paired comparisons using the least squares adjusted means was conducted to examine the differences between the within and between hemispace values. Since only invalid trials were used for this analysis, the overall number of observations per cell was small. Therefore this analysis has limited power and should be considered exploratory.

Results (See Table 4.3)

The multivariate effect for the within and between hemispace comparisons for contralateral targets for the Exogenous Paradigm was not significant $F(5, 10) = 0.70$, $p = .63$, nor was the multivariate effect for the within and between hemispace comparisons for contralateral targets in the Endogenous Paradigm, $F(5, 10) = 1.95$, $p = .17$.

The multivariate effect for within and between hemispace comparisons for ipsilateral targets for the Exogenous Paradigm was not significant, $F(5,10) = 0.99$, $p = .47$, nor was the multivariate effect for the within and between hemispace comparisons for ipsilateral targets in the Endogenous Paradigm, $F(5, 10) = 0.46$, $p = .79$.

Although the MANOVA was not significant, a priori planned comparisons allowed the examination of the univariate effect for each dependent variable. For the contralateral targets in the Exogenous Paradigm, none of the dependent measures were significant (all p values > .05). For the Endogenous Paradigm, only resultant error approached significance, $F(1, 14) = 4.71$, $p = .05$. There was a trend for movements to targets where the cue and target were in the same hemispace to be less accurate than those to targets where the cue and the target were in opposite hemispaces. All other dependent measures were nonsignificant (all p values > .05).

Examining the univariate effect for each dependent variable for the ipsilateral targets in the Exogenous Paradigm, revealed that MT was significant, $F(1, 14) = 5.76$, $p < .05$ and that PV was marginally significant, $F(1, 14) = 3.86$, $p = .07$. No other dependent measures were significant (all p values > .05). As Table 4.3 illustrates, MT was longer and PV slightly lower to targets where the cue and target were in the same hemispace compared to targets where the cue and target were in opposite hemispaces.

Table 4.3

Meridian Effect for Exogenous and Endogenous Targets as a Function of Hemispace of Target (Contralateral vs Ipsilateral)

	RT	MT	PV	%Decel	Error
CONTRALATERAL TARGETS					
Exogenous					
Within	395.3	604.6	89.4	58.3	7.4
SE	10.8	8.7	0.8	0.7	0.7
Between	409.2	607.8	88.5	59.8	7.9
SE	10.6	8.6	0.8	0.7	0.7
Endogenous					
Within	504.1	624.3	88.9	61.5	7.6[a]
SE	10.2	10.2	1.7	1.1	0.5
Between	514.1	620.3	89.4	63.0	6.0[a]
SE	9.9	10.0	1.6	1.1	0.5
IPSILATERAL TARGETS					
Exogenous					
Within	401.0	594.3[b]	94.9[c]	61.7	7.6
SE	9.4	10.9	0.9	0.8	0.4
Between	404.9	557.1[b]	97.7[c]	61.3	7.6
SE	9.6	11.1	0.9	0.8	0.4
Endogenous					
Within	477.8	585.3	93.9	62.4	8.1
SE	11.4	8.8	1.2	0.8	0.5
Between	475.2	603.1	92.5	62.1	8.0
SE	11.2	8.6	1.2	0.8	0.5

[a] indicates a trend toward significance, ($p = .05$).

[b] indicates significance, ($p < .05$).

[c] indicates a trend toward significance ($p = .07$)

DISCUSSION

Constant Accuracy

For both exogenous and endogenous overt movements, resultant error was equivalent across cue validity conditions. Thus, movements to invalidly cued targets had the necessary re-programming completed prior to movement completion in order to ensure the maintenance of accuracy. This finding suggests that, in the current study, even when instructed to complete the movement "as quickly and as accurately as possible", the subjects prioritized the maintenance of accuracy. Indeed, from the perspective of everyday human activities, such a strategy may be the most effective as there is little reward (and even punishment) in our daily lives for reaching inaccurately to rigid surfaces or objects in the environment.

Cuing Effects

As hypothesized, results of the current study indicate that cuing was effective in facilitating performance, for both exogenous and endogenous overt movements. For both target paradigms, consistent with hypotheses, RT was faster, MT was shorter, and PV was higher for movements to validly cued targets compared with movements to invalidly cued targets. It was hypothesized that, given the above results (faster RT, shorter MT, and higher PV for validly cued movements) percent deceleration would be longer for invalidly cued movements compared with validly cued movements, in order to maintain equivalent accuracy. This was not the case. Percent

deceleration and resultant error were equivalent for the valid and invalid conditions. This finding suggests that, although modifications to the motor program took place on-line in the invalid condition (as evidenced by longer MT), these changes were not more likely to occur in the deceleration phase of the movement for invalid than for valid cue conditions. Thus, on the basis of the MT and percent deceleration data one can assume that no greater end-point corrections (i.e., as the finger came closer to intercepting the target) were required in the invalid conditions than in the valid conditions to maintain constant accuracy. Note that this is not to say that a greater amount of changes did not take place for both the valid and invalid cue conditions in the deceleration as compared to the acceleration phase of the movement (as the acceleration and deceleration phases were not compared for each target paradigm), but that the deceleration phases were equivalent in duration for both conditions.

In addition to the findings described above for the cue conditions, there were also differences in the magnitude of the effects of cue condition between the two target paradigms. These magnitude differences revealed subtle variations in the reprogramming of movements (i.e., changes made to the originally preprogrammed movement necessitated by the invalid cues) of the Exogenous and the Endogenous Paradigms.

Exogenous vs Endogenous Overt Movements

There was an overall RT advantage for exogenous overt

movements over endogenous overt movements. This was true both for situations where informational cues were provided (i.e., valid and invalid) and for neutral cue conditions. This advantage was expected, based on previous research literature comparing exogenous and endogenous cues where exogenous cues (somewhat analogous to the exogenous targets used in this study) have been found to elicit faster RT, to be less likely to be ignored, and to "automatically" draw attention even if an individual is attending to another location (Jonides, 1981; Klein, 1993; McCormick, 1997; Muller & Rabbit, 1989; Posner, 1980; Yantis & Jonides, 1984). This exogenous advantage is also consistent with studies which have found abrupt onset targets to automatically elicit a shift of attention (Jonides & Yantis, 1988; Juola, Koshino, & Warner, 1995). Thus, the target presentation itself can be considered to be responsible for the overall RT advantage for exogenous overt movements, in the current experiments, regardless of any effects of shifts of attention or motor programming elicited by the informational cues.

Exogenous Paradigm

The Exogenous Paradigm of the current study was based on the paradigm of Posner et al. (1978) which demonstrated the existence of a covert shift of attention to cued locations prior to target presentation through a simple reaction time response. However, the aiming movement used in this study also required complex motor programming of a visually directed,

multi-joint limb movement through 3-dimensional space. Thus, the present Exogenous Paradigm was thought to incorporate both a covert shift of attention with programming of a complex motor response. The results of the current study revealed that RT was faster, MT shorter, and PV higher for validly cued targets relative to invalidly cued targets. These findings reveal that the facilitation of the movement elicited by a valid cue was not limited to the measure of reaction time but was also noted in certain aspects of the kinematics of the movement. This, in turn, suggests that while some changes to pre-planned motor responses to invalidly cued targets were made prior to movement initiation, further adjustments were made during movement execution. Since the deceleration phase was equivalent for both valid and invalid conditions, even though MT was longer for the invalid than the valid condition, any such on-line modifications of the motor program were not limited to visual feedback-based corrections in the final approach of the target for invalid cues compared with valid cues. The results support the notion that all parameters of the motor program are not specified prior to its initiation (e.g., Rosenbaum, 1980) and that further specification is completed during its execution (e.g., Elliott & Calvert, 1990).

Endogenous Paradigm

The Endogenous Paradigm of the current study was based on that of Rosenbaum and colleagues (Rosenbaum, 1980; Rosenbaum

& Kornblum, 1982) where cued information is thought to allow the programming of movements prior to target presentation. As previously described, this paradigm was considered to involve motor programming only and not a shift of attention (since target information was presented at centre). Results of the Endogenous Paradigm were similar in pattern to those of the Exogenous Paradigm in that RT was faster, MT shorter, and PV higher for validly cued compared with invalidly cued targets, while percent deceleration and resultant error were equivalent. As with the Exogenous Paradigm, valid information regarding the future target position facilitated motor programming and this facilitation was noted for both RT and for the kinematic variables of MT and PV. Thus, it once again appeared that, even in the absence of a presumed shift of visual attention, some changes to an incorrectly preplanned movement were made prior to its initiation, while further changes were made during its execution (e.g., Elliott & Calvert, 1990).

It was of interest to examine the differences in the effects of cuing between the two paradigms in order to make inferences regarding the effects of movements carried out when there was both a shift of attention and motor programming in contrast to when there was motor programming only.

"Costs Plus Benefits"

"Costs plus benefits" (i.e., invalid minus valid condition) was used to examine the differences in the effects

of cuing between the two paradigms. "Costs plus benefits" has been considered to be the most reliable manner for examining cuing effects (Jonides & Mack, 1984; Posner, 1986). The decision not to separate "costs" and "benefits" measures in the current study was based, in part, on the present finding that RT was often slower and MT longer for the neutral cue condition compared with both the valid and invalid cue conditions. As well, in some comparisons in the present study, performance of the neutral cue condition was equivalent to that of either the valid or the invalid cue conditions. This failure to find neutral cue condition performance falling between valid and invalid cue performance has also been noted by others (e.g., Greenwood et al., 1993; Tellinghuisen et al., 1996). Thus, in some situations, the lack of information provided by the neutral cue appears to make it an inherently different task from either of the validly or invalidly cued conditions. This has been discussed at length by other researchers (e.g., Jonides & Mack, 1984; Posner, 1986).

As a result of the lack of separate "costs" and "benefits", only the "costs plus benefits" of the Exogenous and Endogenous Paradigms were compared in an attempt to isolate the effects of shifts of attention from motor programming. If, as hypothesized, there was a shift of attention plus motor programming in the Exogenous Paradigm and motor programming only in the Endogenous Paradigm, then it would be expected that there would be larger "costs plus

benefits" of the Exogenous over the Endogenous Paradigm. This would result from both the extra advantage and the added disadvantage for the valid and invalid cue conditions, respectively, in the Exogenous compared with the Endogenous Paradigm. In contrast to these expectations, however, the "costs plus benefits" for RT, MT, PV and percent deceleration, in the current study, were smaller for the Exogenous Paradigm than for the Endogenous Paradigm. This suggests the possibility that the young subjects in this study may not have shifted attention when presented with the cue in the Exogenous Paradigm but were merely preparing a response (refer to Alternative # 3 on page 49). However, this interpretation is based on the assumption that the preparation of a pointing movement in the two paradigms was equivalent. It is possible that it was not (as was illustrated in the various alternatives of the task analyses outlined in the introduction). It is possible that the young subject group was in some way "more prepared" in the Endogenous Paradigm (compared with the Exogenous Paradigm). Since there were larger "costs plus benefits" for both RT and MT for the Endogenous Paradigm compared with the Exogenous Paradigm, it is also possible that the young subjects actually initiated the movements in the Endogenous Paradigm before they had all the reprogramming completed and therefore relied on more on-line adjustments to complete the movement accurately.

While separate "costs" and "benefits" were not

considered to be a good indication of cuing effects in the current study, it is worth noting that the longer RT for neutral cues compared with invalid cues (for both target paradigms) may suggest that it took longer to program a movement from "scratch" (i.e., neutral cue) than to make changes to an incorrectly planned movement (i.e., invalid cue).

In summary, results of the current study indicate that, as hypothesized, valid cuing provided an advantage (and invalid cuing provided a disadvantage) for both exogenous and endogenous overt movements. This facilitation was evident for RT and for kinematic variables (MT and PV). These results suggest that while some changes to incorrectly preplanned (i.e., invalid) exogenous and endogenous overt movements were conducted prior to movement initiation (resulting in longer RT), further changes were conducted during the execution of the movement (resulting in longer MT and lower PV) in order to maintain equivalent accuracy. Although MT was longer for movements to invalidly cues targets, the proportion of the movement that was conducted during the deceleration phase of the movement did not differ between the valid and invalid conditions. Therefore, there was not a greater requirement to make end-point corrections based on visual feedback in the invalid compared with the valid conditions in order to maintain constant accuracy.

HEMISPACE OF TARGET POSITION

Results of the current study indicate that movements to targets in the ipsilateral hemispace were completed in a shorter amount of time (i.e., shorter MT), had a higher PV, and had a greater percentage of the movement completed in the deceleration phase (i.e., less of the movement completed in the acceleration phase) compared with movements to targets in the contralateral hemispace. The advantage of movements to the ipsilateral hemispace for MT and PV found in the current study are consistent with those noted by previous researchers (e.g., Fisk & Goodale, 1984, 1985; Carey et al., 1996).

Neither of the results for RT noted in the current study using the trigger release or the kinematic data (i.e., equivalent RT to targets in the ipsilateral and contralateral hemispaces; longer RT to targets in the ipsilateral hemispace, respectively) are consistent with those of Fisk and Goodale (1984) or Fisk and Goodale (1985), experiment 1 and experiment 2 (when the eyes were focused at the central fixation point) who found RT was faster to ipsilateral targets. The result of equivalent RT to targets in the ipsilateral and contralateral hemispaces (using the trigger release data) are consistent with those of Fisk and Goodale (1985), experiment 2 using eccentric fixation points where subjects fixated on a position in the same hemispace that the target was presented (as opposed to central fixation). The result of equivalent RT for targets in the ipsilateral and contralateral hemispaces and of

a longer percent deceleration for targets in the ipsilateral hemispace is also consistent with the results of Carey et al., (1996). In experiment 1 of the study of Fisk and Goodale (1985), the percent deceleration of the movements was longer than the percent acceleration of the movements for both ipsilateral and contralateral movements. The findings in the current study of equivalent accuracy for movements to targets in the ipsilateral and contralateral hemispaces is also consistent with the results found by Carey et al. (1996) who had subjects point either to the position that the target was presented to or the mirror symmetrical position of the target. The differences were found to result from the hemispace that the limb reached to and not to the hemispace where the target had been presented.

In the current study, since PV was higher (and MT shorter) for movements to the ipsilateral hemispace compared with those to the contralateral hemispace, it is likely that subjects had to begin to decelerate early (during movements to the ipsilateral hemispace) in order to avoid hitting the computer screen with too much force. For movements to the contralateral hemispace, where the PV was lower and the MT longer, there may have been less need to decelerate early as adjustments could be conducted more smoothly over the complete trajectory.

In the present study, the difference in the results for RT using the trigger release (i.e., RT equivalent for

ipsilateral and contralateral reaches) and the kinematic data (i.e., longer RT to ipsilateral compared with contralateral targets) suggests that the subjects in this study began movements to contralateral targets by lifting the knuckle relatively earlier and began movements to ipsilateral targets by lifting the finger relatively earlier. On average, however, knuckle movement consistently preceded finger movement.

In summary, there is evidence for an effect of MT, PV, and percent deceleration in the current study. The results suggest that these effects were likely due to the constraints imposed by biomechanical factors related to joint and muscle function (Carey et al., 1996; Fisk & Goodale, 1985).

MERIDIAN EFFECTS

A "meridian effect" is present when RT is longer in the situation where a precue and a target are presented in opposite hemifields (either vertically or horizontally), compared to the situation where they are presented within the same quadrant. This effect has been noted in the RT literature using endogenous precues and exogenous targets (Reuter-Lorenz & Fendrich, 1992; Rizzolatti, Riggio, Dascola, & Umilta, 1987; Umilta, Riggio, Dascola, & Rizzolatti, 1991) and to be absent in those paradigms pairing exogenous precues with exogenous targets (Reuter-Lorenz & Fendrich, 1992; Umilta et al., 1991).

The meridian effect has been considered to provide evidence that covert shifts of visual attention and overt eye movements are under the control of the same mechanism (e.g.,

Rizzolatti, Riggio, Dascola, & Umilta, 1987). Given that only the Exogenous Paradigm in the current study was considered to involve a shift of attention, but that a similar effect might exist for motor programming of directed limb movements (Rizzolatti et al., 1987; Sheliga et al., 1997) this effect was examined separately in both the Exogenous and the Endogenous Paradigms (both of which involve motor programming but only the former involves an attentional shift). As well, given the effects of the hemispace of target position found, it was necessary to examine the meridian effect for ipsilateral and contralateral targets separately.

As hypothesized, there was no meridian effect for the Endogenous Paradigm for either ipsilateral or contralateral targets. Although there was a trend ($p = .05$) for resultant error to be slightly greater for targets where the cue and target were presented in the same hemispace for contralateral targets, in the absence of any significant differences in the other dependent variables, this finding is considered coincidental and irrelevant.

In the Exogenous Paradigm, there were no differences in the dependent measures for contralateral targets as a function of crossing the meridian. For ipsilateral targets, there was no difference in RT (which has been considered the hallmark of the meridian effect). There was a significant difference in MT (and a trend for significance for PV) as a function of crossing the meridian. However, the direction of this

difference was opposite what would be expected given that crossing the meridian is associated with a disadvantage. That is to say that MT was longer (and the PV slightly lower) when the cue and target were presented in the same hemispace compared to when they were presented in opposite hemispaces. Had this result been related to crossing the meridian (as described in the literature), one would have expected that MT would have been shorter instead of longer when one did not have to cross the meridian. It is not clear why the current result is seen with ipsilateral and not contralateral movements. There may be biomechanical aspects of reaching across the body midline that negate any effects for contralateral movements. This has not yet been noted in the literature on meridian effects and may reflect the fact that these paradigms have been limited to measures of RT and have not used complex movements.

The absence of a meridian effect for RT in the in the Exogenous Paradigm of the current study may have been due to differences in the experimental design. Previous studies have examined a simple manual movement (button press) or a saccadic eye movement. As well, most of the previous studies used an apparatus to hold the head in a stable position (Reuter-Lorenz & Fendrich, 1992; Sheliga et al., 1994; Umilta et al., 1991). The current study was interested in examining complex movements that would closely approximate everyday movements (where the head is generally free to move). It is also

important to note that the current study had a small sample of observations available to examine the meridian effect. Previous studies have used a greater number of trials and often had subjects perform blocks of trials on a number of different days. It is possible that the differences noted here result from these differences in the paradigms used or the smaller number of observations available for analysis in this study which reduced "power" to detect true differences.

CONCLUSIONS

Constant accuracy appeared to be the goal the subjects used in this study, even though they were asked to make movements with both speed and accuracy. There was an advantage to being provided valid informational precues for both exogenous and endogenous overt movements and a disadvantage to being provided with incorrect information. These advantages were not limited to the RT interval (as has been the focus of most studies) but were also evident in the kinematic parameters (e.g., MT, PV) of the movement trajectory and were noted in both the Exogenous and Endogenous Paradigms.

For the young healthy adults used in this study, motor programming for exogenous and endogenous targets had a similar pattern of results. Exogenous overt movements had a greater overall RT advantage compared with endogenous overt movements but had a smaller magnitude of advantages/disadvantages (i.e., "costs plus benefits") as a result of informational precues.

Although part of the overall RT advantage noted in the

Exogenous Paradigm may be attributed to the exogenous manner of the target presentation, the MT and PV advantage cannot be attributed to this factor (i.e., the MT and PV of the neutral conditions for the two paradigms were equivalent). Therefore, the MT and PV advantage of the Exogenous Paradigm (over the Endogenous Paradigm) are interpreted as an indication that there is a facilitation of motor programming provided by exogenous target presentation.

The effects of hemispace of target position noted in the current study are similar to previous research that found shorter MT and higher PV for ipsilateral movements (Fisk & Goodale, 1985), longer percent deceleration for ipsilateral movements (Carey et. al., 1996) and equivalent RT and accuracy for ipsilateral and contralateral movements (Carey et al., 1996). As well, the laterality analysis reflected the different pattern of muscle activation for the initiation of movements to ipsilateral versus contralateral targets (based on the differences noted between the finger and knuckle IRED's). Although the knuckle movement typically preceded the finger movement, this difference was greater for contralateral movements. This likely reflects the increased proximal muscle activation required for movements that cross the body midline.

There was no traditional meridian effect (i.e., longer RT when crossing the meridian) found in this study. The effects noted for the meridian analysis (i.e., longer MT and lower PV for movements that did not cross the meridian) do not

provide support for there being a disadvantage for meridian crossing. Thus, the effects noted in the current study provide support for those who postulate that covert shifts of attention and eye movements (or motor programs generally) are separate functions controlled by different mechanisms (e.g., Klein and colleagues). However, it must be noted that there were few observations available for this analysis resulting in low "power" to detect true effects.

Results of the current study indicate that there is an advantage to using kinematic measures in addition to RT, in the integration of attention and motor function perspectives, and to using complex movements (e.g., pointing, reaching) rather than simple movements (button press or release) when investigating visuomotor integration. The design and results of the current study revealed the complex interaction of attention and motor programming that must exist in everyday functioning.

In the following chapter, the effects of covert shifts of attention on motor programming are examined in healthy elderly subjects. The same two paradigms that were used for the healthy young subjects were used once again. Results from the performance of the healthy elderly are discussed first and then compared with those from the healthy young subjects in order to examine the effects of aging on the integration of attention and motor programming. Hemispace of target position and meridian effects are also examined.

Chapter 5 - Study 2

THE EFFECTS OF VISUAL ATTENTION AND MOTOR PROGRAMMING ON MANUAL AIMING MOVEMENTS IN HEALTHY ELDERLY ADULTS

INTRODUCTION

It is commonly thought that many of our capacities decrease in effectiveness or efficiency as we age. This is particularly true of the speed of both cognitive and motor functions (see Salthouse, 1985 for a review). Although various hypotheses have been postulated to explain this slowing, they generally fall into two main types. The first attributes the slowing to the neuroanatomical changes associated with aging (e.g., loss of neurons, decreased blood flow), while the second attributes the slowing to a change in strategies adopted by older individuals (e.g., more cautious approaches with a greater focus on accuracy) (see Roy, Weir, & Leavitt, 1996 for a review). These two hypotheses are not mutually exclusive.

The following chapter is devoted to examining the changes in visual attention and motor programming that occur with healthy aging. It will begin by briefly addressing the physiological changes associated with the healthy aging process. Next, the literature on visual attention and on motor function in healthy aging will be reviewed. The remainder of the chapter will be devoted to presenting the details of the current study and a discussion of the results. The same paradigm and methodology that was used for the healthy young

subjects was administered to the healthy elderly subjects.

Physiological Changes Of Aging

Changes that occur to some degree in the central nervous and motor systems of humans with the aging process include: decreased number of neurons and synapses, dendritic regression, reduced dopaminergic function in the basal ganglia, and decreased number and size of muscle fibres (Mortimer, Pirozzolo, & Maletta, 1982). These age-related changes have been considered to contribute to a reduction in information processing speed and to an increase in reaction time. A reduction in the number and size of muscle fibers and motor neurons has been considered to result in reduced muscle strength and decreased speed of muscular contractions (Welford, 1982). These changes have also been associated with longer movement durations (e.g., Roy et al., 1993). Other age-related changes include disturbances in the temporal organization of movements, reduced speed of performance of sequential repetitive movements, and increased performance deficits in complex motor tasks (Welford, 1982).

Visual Attention and Aging

Research on visuospatial attention in aging has used numerous types of paradigms and has provided results that are somewhat inconclusive. For the most part, research suggests that the visuospatial attention system is relatively unaffected by the healthy aging process. This conclusion is based on results of studies which reveal that healthy elderly

adults are able to use cues to improve performance (eg., reduce RT) in a manner similar to that of healthy young adults (e.g., Hartley, Kieley, & Slabach, 1990, experiment 1; Hartley et al., 1992; Robin & Rizzo, 1992; Tellinghuisen, Zimba, & Robin, 1996). An exception to this was found in a study by Folk & Hoyer (1992), experiment 2, where elderly subjects were unable to use cued information concerning the future target to facilitate performance (i.e., valid RT = invalid RT). The authors concluded that the elderly subjects required a longer time to interpret endogenous cues compared with young subjects.

Despite the general findings that elderly subjects are able to use cues to facilitate performance, there have nevertheless been some performance differences between young and elderly adults that have been associated with the aging process. For example, elderly subjects have generally been found to have larger "costs plus benefits" (i.e., invalid RT minus valid RT) compared with young subjects (e.g., Folk & Hoyer, 1992, experiment 3; Hartley et al., 1990; Nissen & Corkin, 1985). The larger "costs plus benefits" (or larger "costs" alone) of elderly subjects compared with young subjects has led to the conclusion that there may be a slowing of the attentional disengagement function in the healthy elderly (for review, see D'Aloisio & Klein, 1990).

These consistent differences in the "costs plus benefits" between elderly and young subjects have been found, by some,

to be related to the SOA. For example, Greenwood et al. (1993), using a letter discrimination task, noted that elderly subjects had larger "costs plus benefits" at a 500ms SOA and showed a trend toward having larger "costs plus benefits" at a 2000ms SOA, but had equivalent "costs plus benefits" to young subjects at the 200ms SOA. These authors also found that the neutral condition often elicited longer RT's than the invalid conditions leading them to suggest that the neutral may not have been truly "neutral" in their paradigm. Brodeur and Enns (1997) also used a letter discrimination task and found that the young subjects used in their study had significant "costs plus benefits" at all SOA's (200ms, 400ms, 600ms, 800ms) while the elderly subjects had "costs plus benefits" only at the 800ms SOA. They interpreted this to indicate that the elderly subjects required a longer period of time to make use of the cues than did young subjects. This interpretation is consistent with that of Folk and Hoyer (1992). However, Hartley et al. (1990) found no differences in the time course of the appearance of "costs plus benefits" between young and elderly subjects (i.e., both groups had "costs plus benefits" at 300ms and 500ms SOA's). Hartley et al. (1990) presented each of the SOA's in different blocks while Folk and Hoyer (1992) and Bodeur and Enns (1997) interspersed the various SOA's within trial blocks.

In summary, the research on visual attention and aging has generally noted that healthy elderly adults are able to

use cues to facilitate performance. The elderly have also generally been noted to have larger "costs plus benefits" compared with the young (for endogenous cue conditions). The differences in "costs plus benefits" between elderly and young subjects have been associated, by some researchers, with the SOA used while others have found no relation between the two (Hartley et al., 1990). Those who have associated differences in "costs plus benefits" between elderly and young subjects with SOA, have suggested that the elderly may require a longer time to process information compared with healthy young adults (e.g., Brodeur & Enns, 1997; Folk & Hoyer, 1992; Greenwood et. al., 1993).

MOTOR FUNCTION AND AGING

The literature abounds with studies examining motor functioning and aging. This research encompasses numerous perspectives and uses many different types of paradigms. The following section will focus on cuing paradigms which have investigated the effects of healthy aging on motor programming, including kinematic studies of motor programming (using hand or finger movements), as these studies are more directly relevant to the current study.

For the most part, the literature on motor programming in aging generally describes a slowing of RT, MT, and a lowering of PV compared with young adults (e.g., Goggin, Stelmach & Amrhein, 1989; Gottsdanker, 1980a,b; Stelmach, Goggin, & Amrhein, 1988; Stelmach, Goggin, & Garcia-Colera, 1987; Roy,

Winchester, Weir, & Black, 1993). However, aging does not appear to affect the ability to use advance information about future targets to preprogram movements (e.g., Gottsdanker 1980a,b; Stelmach et al., 1987, 1988). Nevertheless, some research has revealed that the RT but not MT differences between young and elderly adults increase when a fewer number of parameters are cued (e.g., arm or direction or extent versus arm, direction and extent) (e.g., Stelmach et al., 1987). Similar to some of the visual attention literature, these results have been interpreted to indicate that elderly subjects require more time to use cued information than do young subjects.

Kinematic studies have attributed longer overall MT for elderly subjects to either longer deceleration phases only (e.g., Roy et al., 1993) or to both longer acceleration and deceleration phases (e.g., Pratt, Chasteen, & Abrams, 1994) compared with young adults. Some research has associated longer MT for elderly subjects with a deficit in the ability to process feedback (Larish & Stelmach, 1982; Stelmach et al., 1987; Warabi, Noda, & Kato, 1986) while others have associated it with a deficit in the ability to generate force and to control force generation (Goggin & Stelmach, 1990; Pratt et al., 1994; Stelmach, Goggin & Amrhein, 1988). Roy et al. (1993) has suggested that a deficit in force generation is a particular problem for elderly subjects, at the time of movement initiation. In contrast to these findings, Walker,

Philbin, and Fisk (1997) have argued that elderly adults are able to produce equivalent force to that of young adults but that they choose not to do so (i.e., they use a more conservative approach than young adults). In support of this hypothesis, they cite results of their research where elderly subjects made more small corrective submovements when positioning a cursor inside target boxes of varying sizes in comparison with young subjects.

In a similar vein, Larish and Stelmach (1982) suggested that elderly adults may be more deliberate in deciding upon and choosing the appropriate response prior to initiating movements in comparison with young adults. Results of research examining accuracy of performance for young and elderly subject groups is inconclusive. Elderly subjects have been found to be more accurate than young subjects (e.g., Larish & Stelmach, 1982), to have more errors than young subjects (e.g., Stelmach et al., 1988), or to have equivalent accuracy to young subjects (e.g., Hartley et al., 1990, experiment 1; Smith & Brewer, 1995; Stelmach, Amrhein, & Goggin, 1988). Although both RT and MT have been noted to be increased in elderly adults compared with young adults, it has been suggested that there are greater differences in RT between young and elderly subjects than there are in MT (e.g., Warabi et al., 1986).

In summary, the above literature suggests that, generally, RT and MT are slower and PV lower in elderly adults

compared with young adults. However, elderly subjects have been shown to be able to use cues in a similar manner to the young to preprogram movements. There have been differences noted in the trajectory of the movements of young and elderly adults (e.g., differences in acceleration time, deceleration time, and the number subcomponents or corrective movements made). Whether these changes result in greater accuracy is inconclusive. These results (particularly those of kinematic analyses) suggest that there may be differences in movement programming that show up as differences in the movement trajectory between elderly and young adults, and that changes in motor functioning with aging may not be fully explained by the widely accepted notion of a general slowing of reaction time for elderly adults compared with young adults.

It has generally been the case that the literature on motor programming and visual attention in aging have been conducted in isolation from one another. However, one study investigating motor programming attempted to incorporate an analysis of the effects of shifts of visual attention on motor programming. This study was conducted by Amrhein, Von Dras, and Anderson (1993). They used a movement plan restructuring task (based on Rosenbaum & Kornblum, 1982) where valid or invalid cues for arm or direction or arm and direction were provided. A four-light, two-column visual display and a corresponding response panel were used. Trials began with the subject pressing the home keys with both hands, followed by

the 200ms presentation of the precue. This was followed by variable preparatory intervals (250ms, 500ms, 1000ms and 2000ms) and then by target presentation. Upon target presentation, the subject was to release the home key appropriate to the lateral position of the target (i.e., left or right hand) and press the corresponding target key according to its vertical position (i.e., up or down). Results indicated that both elderly and young subjects were able to use advance cues to preprogram a movement (i.e., RT shorter to validly cued targets compared with invalidly cued targets). However, at the 1000ms and 2000ms preparation interval, the elderly subjects had faster RT for reprogramming direction than for reprogramming arm alone or for arm and direction (which were equivalent). There were no differences in MT as a function of preparatory interval for elderly subjects and no RT or MT differences as a function of preparatory interval for the young subjects. The authors concluded that by the 1000ms SOA, elderly subjects had "lost" the preparation for direction (which accounted for the faster RT for reprogramming direction compared with arm or arm and direction) and that the "loss" was confined to the RT interval and was permanent (since it remained at 2000ms). To examine the possible confound of movement reprogramming with the reorienting of visual attention, the authors used the same paradigm in a second experiment but only required the subject to release the home key when the target was presented and not to move the finger

to the target key. Results indicated that in the valid conditions (i.e., where it was considered there was no shift of attention from cue to target presentation), that RT was faster than on the invalid conditions, which were considered to be composed of either a lateral shift of attention (arm change from cued arm), a vertical shift of attention (direction change from cued direction), or a diagonal shift of attention (arm and direction change). Results indicated that there were no effects of preparatory interval or of attentional shift type (i.e., any of the invalid cues) on any of the reprogramming parameters that had been noted on the movement restructuring task (i.e., differences in the preparation of direction alone compared to arm alone or arm and direction at the 1000ms SOA and the 2000ms SOA for the elderly). This was interpreted by the authors, to indicate that the reprogramming changes revealed in the first experiment were not due to age related differences in the orienting of attention but to those associated with movement programming.

The Amrhein et al. (1993) study represents an advance in the investigation of motor programming and visual attention in elderly subjects in that it attempted to separate the effects of motor programming from shifts of attention (by attempting to remove the need to preprogram a movement of the finger). For this reason, it is particularly relevant to the current study which also separated the effects of motor programming

from shifts of visual attention. However, Amrhein et al.'s (1993) study differs from the current study in that it used a simple button press response. It also differs in that it used a fairly complex analysis of shifts of attention in that it examined three types of "orienting shifts" on invalidly cued trials (i.e., lateral, diagonal, and vertical) in comparison to no "orienting shifts" on validly cued trials. As well, it incorporated four different preparatory intervals between cue and target. Of most relevance to the current study was the fact that the authors attributed the changes noted in the movement restructuring task as a function of aging to be due to age-related differences in movement restructuring and not to age-related effects of reorienting of visual attention.

In summary, the literature reviewed above has provided an overview of the differences involved in movements of healthy elderly adults (e.g., slower RT, longer MT, lower PV, longer deceleration times, variations in subcomponents of the movements). One study to date has examined the potential confound of shifts of visual attention and movement restructuring. Most of the studies reviewed used the manipulation of an object (e.g., computer mouse, pen, stylus, rotational levers) rather than a pointing movement to a target with the finger. Those that used a button press with the finger did not use a kinematic analysis. While Amrhein et al. (1993) examined shifts of attention and movement restructuring, this analysis was limited to RT and MT and used

a button press response. Goggin and Stelmach (1990) combined visual cues with a kinematic analysis of motor programming but they did not examine the effects of shifts of visual attention. The following study combined covert shifts of visual attention and advance information on pointing movements (using a three dimensional aiming finger movement) and the effects of aging.

HYPOTHESES - ELDERLY SUBJECT GROUP

Cue Condition

For the elderly subject group, a number of hypotheses regarding cuing effects were developed based on the literature of visual attention, motor programming and kinematic analyses as well as the results of the young subject group used in this thesis. They were as follows:

Informational cues that provide valid information were expected to provide an advantage of movements for RT, MT, and PV for both the Exogenous and Endogenous Paradigms (i.e., valid conditions were expected to have an advantage over invalid conditions). Based on the results of the young subject group, the percentage of the movement that took place in the deceleration phase of the movement and the overall accuracy were expected to be equivalent in the valid and invalid cue conditions of both paradigms. As was hypothesized (and found) for the young subject group, RT was expected to be faster for the Exogenous Paradigm compared with the Endogenous Paradigm. Based on the results of the young subject group, the neutral

condition was not expected to fall between the valid and invalid conditions.

Between Group Effects

The elderly subject group was expected to have slower RT, longer MT, and lower PV than the young subject group. If the elderly subject group had a greater focus on accuracy than the young subject group (as some literature suggests), they were expected to have a longer percentage of the movement conducted during the deceleration phase than the young subject group and a resulting greater accuracy than the young subject group.

The elderly subject group was expected to have larger RT "costs plus benefits" than the young subject group. Since, in the literature, this effect has been found in studies examining covert shifts of attention, the larger RT "costs plus benefits" were expected in the Exogenous Paradigm. However, since it has also been suggested in the literature that elderly individuals may take longer to use information compared with young individuals, it was possible that the elderly subject group would have larger RT "costs plus benefits" compared with the young subject group in the Endogenous Paradigm as well (i.e., they would take longer to make changes when presented with a cue that was determined to be invalid).

Hemispace of Target Position Effect

The pattern of results for the elderly subject group was expected to be equivalent to those of the young subject group,

since there has been no research finding laterality differences based on age. Thus, it was hypothesized that: MT would be shorter and PV higher for movements to targets in the ipsilateral compared with the contralateral hemispace. Percent deceleration was expected to be longer for ipsilateral compared with contralateral movements and resultant error was expected to be equivalent for ipsilateral and contralateral targets.

Meridian Effect

There was no traditional meridian effect for the young subject group (i.e., RT faster when cue and target were in the same hemifield compared to when they were in the opposite hemifield). The fact that the young subject group may not have shifted attention may have accounted for this. If the elderly subject group shifted attention in the Exogenous Paradigm, it was hypothesized that there would be a meridian effect for RT in the Exogenous Paradigm. Based on the results of the young subject group, no meridian effect was expected for the Endogenous Paradigm.

METHODS

Subjects

Subjects were 15 healthy elderly adults (13 females; 2 males) who were recruited from an ongoing aging study (Canadian Study of Health and Aging) and from community volunteers. There was a significant difference in gender between the young and the elderly subject group (chi square = 3.96, $p < .05$) with the elderly subject group having a greater number of females and a fewer number of males than the young subject group (8 females, 7 males). Mean age for the elderly subject group was 69.5 (SD = 5.3) with a range of 58 to 77 years. Mean level of education was 13.4 years (SD = 3.2) with a range of 6 to 18 years. The education level of the elderly subjects was significantly less than that of the young subjects ($p < .05$) whose mean level of education was 17.6 years (sd = 1.8 years) with a range of 13 to 20 years. Informed consent was obtained from all subjects in accordance with the procedures approved by the Queen Elizabeth II Health Sciences Centre Research Ethics Committee.

It is noteworthy that the majority of the elderly subjects used in this study were females. This was not intentional but, rather, was the result of the gender of the participants available. In order to determine that no differences between young and elderly subjects were due to the greater proportion of females in the elderly sample, a MANOVA was conducted on the young subject group using gender as a

variable. No significant multivariate main effects of gender or interactions were significant. These results were as follows: gender, $F(6, 8) = 1.82$, $p = .21$; gender by condition, $F(12, 44) = 1.66$, $p = .11$; gender by target type, $F(6, 8) = 1.06$, $p = .46$; gender by condition by target type, $F(12, 44) = 0.90$, $p = .55$. Because of these results and the fact that there are no indications in the literature of gender differences (e.g., Gottsdanker 1980b noted no gender differences) in either attentional allocation or the kinematics of aiming movements, gender was not included as a variable in further analyses.

Procedure

The only differences from the procedure described in Chapter 3 were that there were a greater number of practice trials (maximum of an additional 20 trials) required for some of the elderly subjects to learn the task. In addition, only two blocks of 60 experimental trials were presented (rather than the three blocks presented to the young subjects). Although this reduced the power of the analyses, it was deemed necessary in order to avoid introducing the effects of fatigue into the study.

Analyses

The same procedure for statistical analyses were followed as were described in the previous chapter. Once again, Multivariate Repeated Measures Analysis Of Variance (MANOVA) were followed up with univariate ANOVA's. In all tests of

significance an alpha level of 0.05 was adopted. All planned comparisons were conduced using the least squares adjusted means.

Five analyses were conducted on the data for the elderly subjects. Analysis 1 examined the effect of cue validity on the dependent variables in the elderly subject group. Analysis 2 compared the effects of cue condition of the elderly group with the young subject group. Analysis 3 examined the laterality effect and compared these results with those of the young. Analysis 4 examined the meridian effect of the elderly subject group alone while Analysis 5 examined the meridian effect of both the young and healthy elderly subject groups combined. This latter analysis was designed to improve the power of the meridian effect analysis by increasing the number of trials available.

ANALYSIS 1

ELDERLY CONTROL GROUP - CUE CONDITION

Analysis 1 was a 3 (Cue Condition - Valid, Invalid, Neutral) X 2 (Target Type - Endogenous, Exogenous) Multivariate Repeated Measures Analysis of Variance (MANOVA) using a SAS General Linear Models Procedure. Cue condition and target type were within subject variables. Analysis 1 included only the elderly control subjects. Dependent measures were reaction time (RT), Movement duration time (MT), peak velocity (PV), percent deceleration, and resultant error. A series of a priori planned paired comparisons using the least squares

adjusted means was conducted to examine the differences between target types and among cue conditions.

Results

The multivariate MANOVA revealed multivariate main effects of cue condition, $F(10, 50), = 3.07, p < .005$. There was a marginal effect of target type, $F(5, 10), = 2.81, p = .07$, and no multivariate cue condition by target type interaction $F(10, 50), = 1.32, p = .25$. The significance of the effects in the multivariate analysis allowed the inspection of the univariate Analysis of Variance (ANOVA) for each of the five dependent variables.

Reaction Time (See Figure 5.1)

There was a univariate main effect of cue condition for RT, $F(2, 28) = 20.08, p < .0001$. Comparison of the least squares adjusted means (collapsing across target type) revealed that the mean RT for the valid condition (M = 562.3ms, SE = 9.5ms) was significantly faster than the invalid condition (M = 644.7ms, SE = 16.4ms; $t = 4.35, p < .0005$) and the neutral condition (M = 667.3ms, SE = 16.3ms; $t = 5.54, p < .0001$). The RT for the neutral and the invalid conditions did not differ significantly from each other ($t = 0.97, p = .33$). There was a univariate main effect of target type, $F(1, 14) = 15.73, p <.002$. Comparison of the least squares adjusted means (collapsing across cue condition) revealed that the mean RT for the Exogenous Paradigm (M = 586.2, SE = 13.7) was significantly faster than the Endogenous Paradigm (M =

663.3ms, *SE* = 13.7; *t* 3.96, *p* < .001). There was a marginal cue condition by target type interaction, *F*(2, 28) = 2.58, *p* = .09. Examining each of the cue conditions within the two target types revealed that all cue conditions of the Exogenous Paradigm were significantly faster than those of the Endogenous Paradigm. This interaction is explained by the fact that, while the pattern of results for the two target types was similar, the "costs plus benefits" (i.e., invalid RT minus valid RT) for the Exogenous Paradigm (101.5ms; *t* = 7.39, *p* < .0001) were larger than those of the Endogenous Paradigm (63.4ms; *t* = 4.63, *p* < .0001). The difference in the "costs plus benefits" of the two target paradigms was marginally significant (*p* = .06).

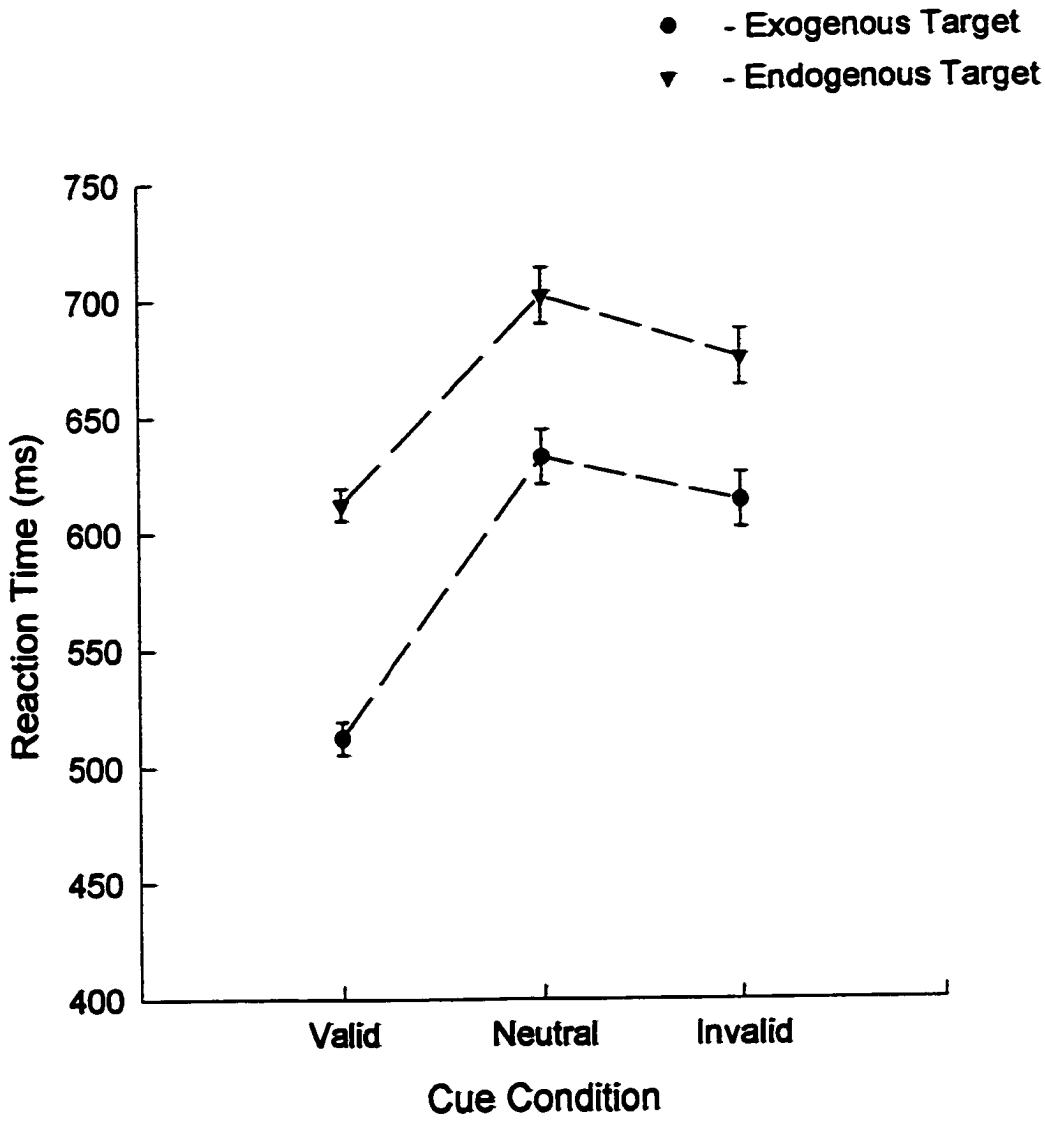


Figure 5.1 - Reaction time as a function of cue condition and target paradigm. The error bars represent the standard error of the least squares means.

Movement Duration Time (See Figure 5.2)

There was a marginal main effect of cue condition for MT, $F(2, 28) = 28.01$, $p = .08$. Comparison of the least squares adjusted means (collapsing across target type) revealed that the mean MT for the valid condition (M = 728.8ms, SE = 2.2ms) was significantly faster than the neutral condition (M = 738.5ms, SE = 3.9ms; $t = 2.17$, $p < .05$). The valid and invalid conditions did not differ significantly from each other (t = 1.45, $p = .15$) nor did the invalid and neutral conditions (t = 0.59, $p = .55$). There was no univariate main effect of target type, $F(1, 14) = 1.30$, $p = .27$ and no cue condition by target type interaction, $F(2, 28) = 0.31$, $p = .74$.

The "costs plus benefits" for the Exogenous Paradigm (3.8ms) were not significant ($t = 0.76$, $p = .45$) and those for the Endogenous Paradigm (9.2ms) were only of marginal significance ($t = 1.86$, $p = .07$).

Peak Velocity (See Figure 5.3)

There was no univariate main effect of cue condition for PV, $F(2, 28) = 1.52$, $p = .23$ or target type, $F(1, 14) = 2.72$, $p = .12$ and no cue condition by target type interaction, $F(2, 28) = 2.41$, $p = .11$. Average PV ranged from 71.6cm/sec to 75.2cm/sec across subjects and target types.

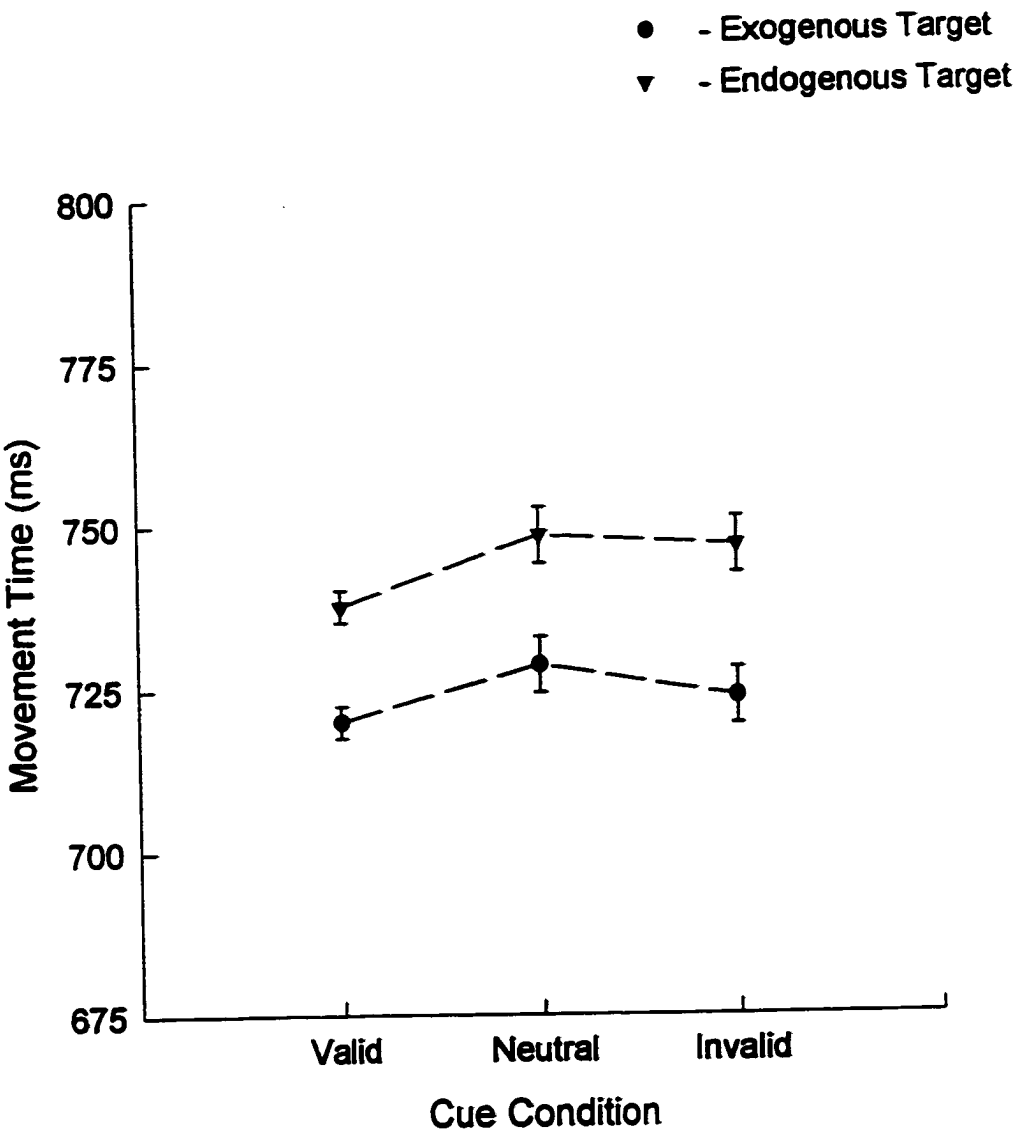


Figure 5.2 - Movement time as a function of cue condition and target paradigm. The error bars represent the standard error of the least squares means.

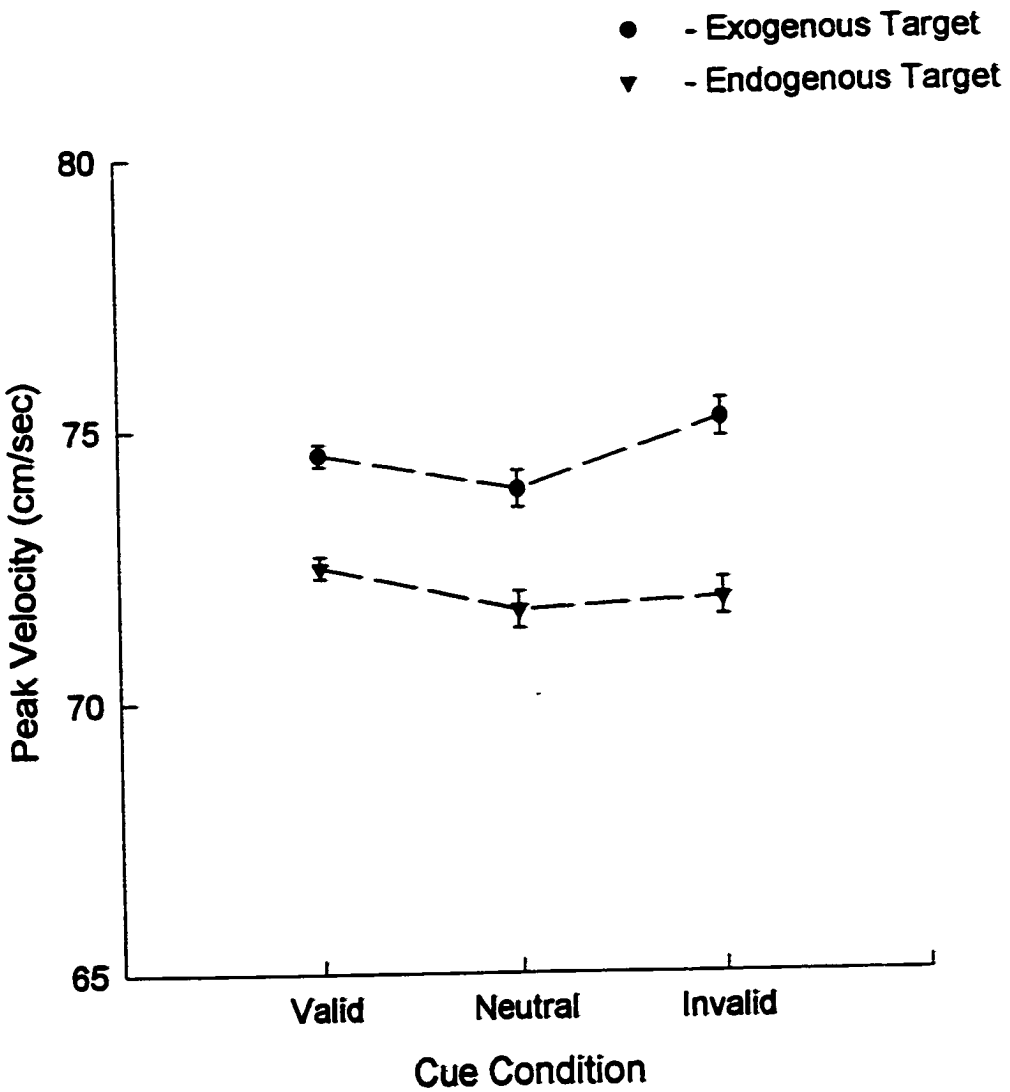


Figure 5.3 - Peak velocity as a function of cue condition and target paradigm. The error bars represent the standard error of the least squares means.

Percent Deceleration (See Figure 5.4)

There was no univariate main effect of cue condition for percent deceleration, $F(2, 28) = 1.85$, $p = .18$, target type, $F(1, 14) = 0.89$, $p = .36$, and no cue condition by target type interaction, $F(2, 28) = 1.15$, $p = .32$. Average percent deceleration ranged from 59.1% to 60.4% across subjects and target types. A priori planned comparisons revealed significant "costs plus benefits" for the Exogenous Paradigm (1.0%, $t = 2.48$, $p < .02$) while there were none for the Endogenous Paradigm (0.26%; $t = 0.59$, $p = .55$).

Resultant Error (See Figure 5.5)

There was no univariate main effect of cue condition for resultant error, $F(2, 28) = 0.34$, $p = .71$, target type, $F(1, 14) = 0.10$, $p = .75$, and no cue condition by target type interaction, $F(2, 28) = 0.49$, $p = .61$. Average error ranged from 8.1mm to 8.5mm among subjects and across both target types.

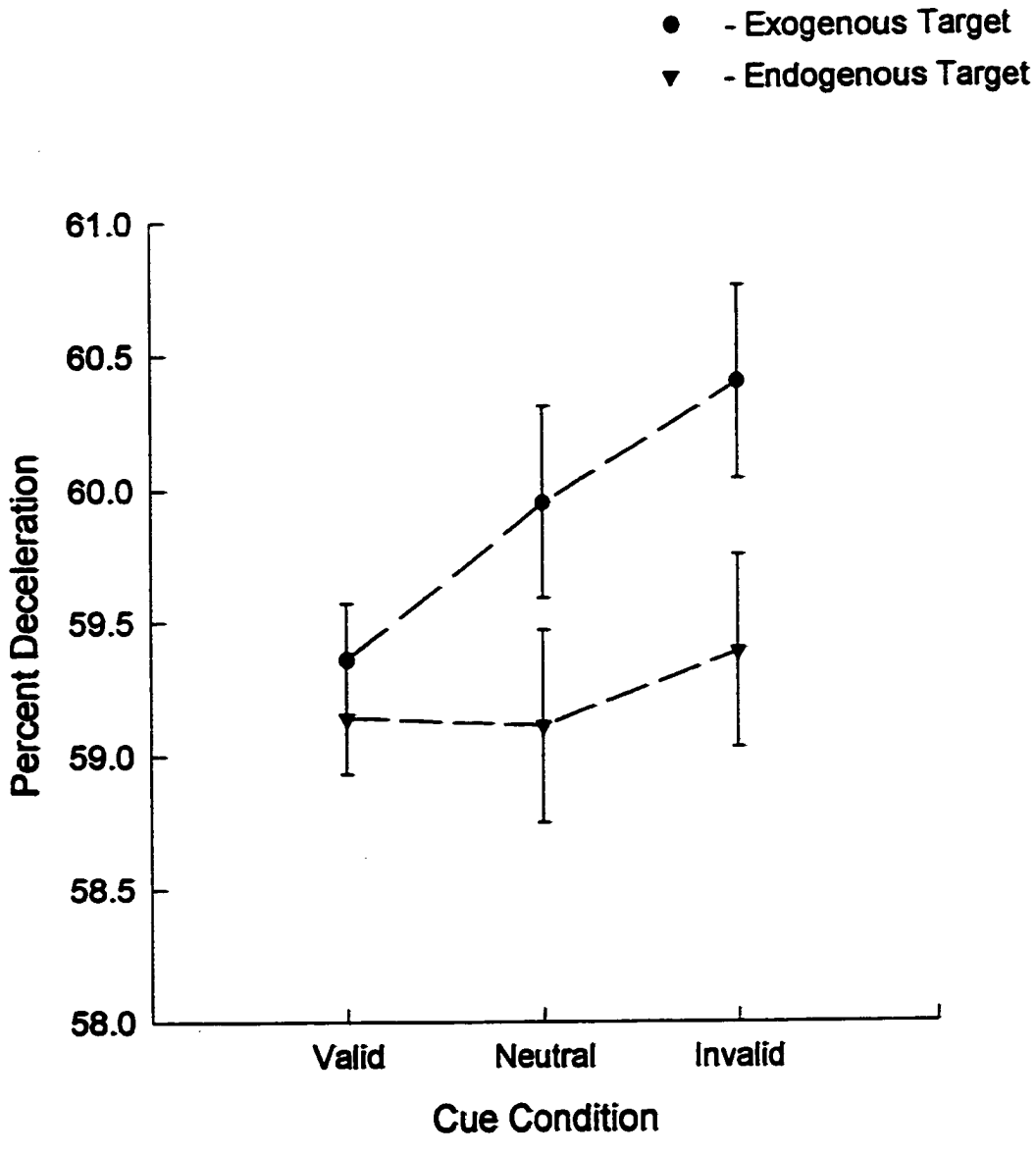


Figure 5.4 - Percent deceleration as a function of cue condition and target paradigm. The error bars represent the standard error of the least squares means.

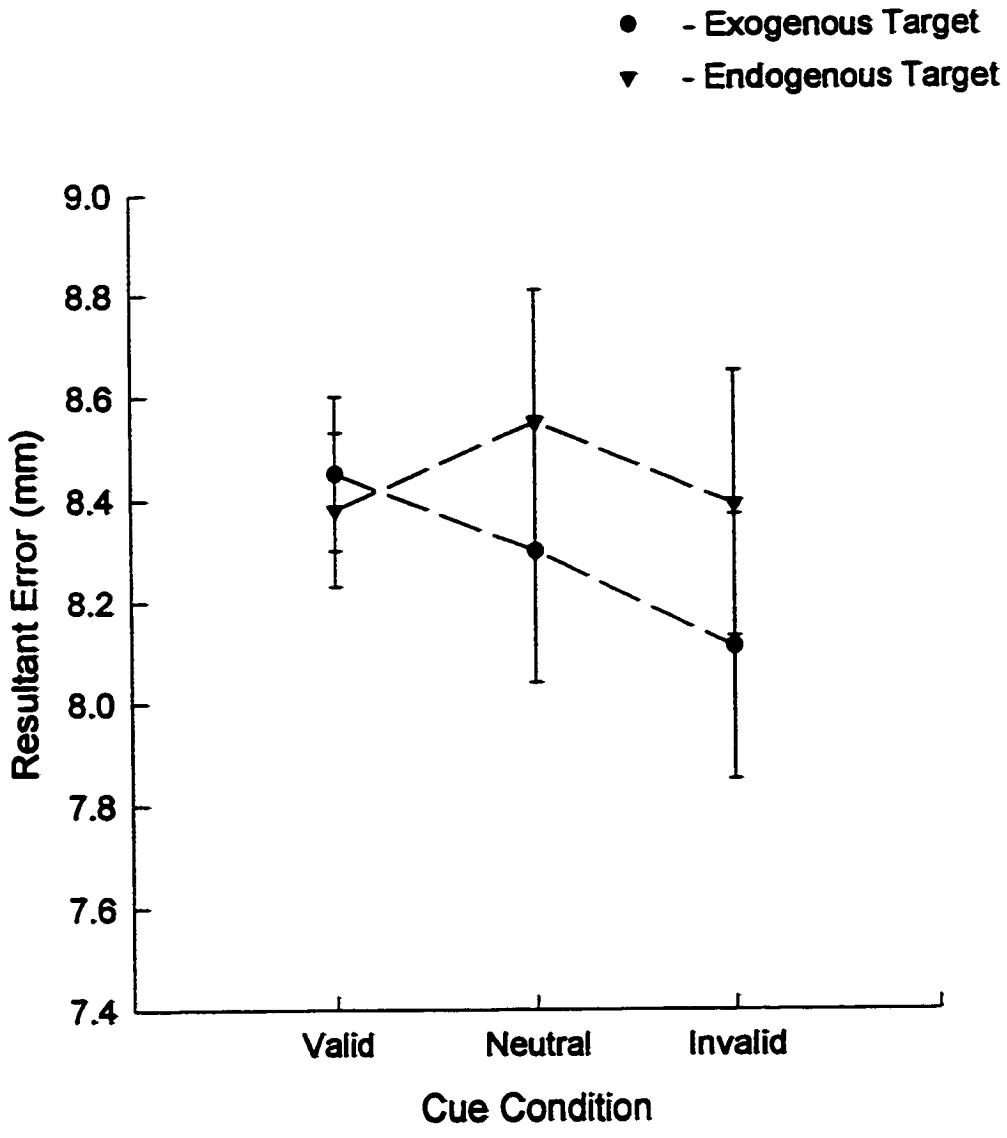


Figure 5.5 - Resultant error as a function of cue condition and target paradigm. The error bars represent the standard error of the least squares means.

ANALYSIS 2

ELDERLY VERSUS YOUNG SUBJECT GROUPS - CUE CONDITION

Analysis 2 was a 3 (Cue Condition - Valid, Invalid, Neutral) x 2 (Target Type - Endogenous, Exogenous) x 2 (Age Group - Young, Elderly) Multivariate Repeated Measures Analysis of Variance (MANOVA) using a SAS General Linear Models procedure. Group was a between subject factor and cue condition and target type were within subject factors. Dependent measures were reaction time (RT), movement time (MT), peak velocity (PV), percent deceleration, and resultant error. A series of planned paired comparisons using the least squares adjusted means was conducted to examine the differences between target types and among cue conditions for the two age groups.

Results

The multivariate MANOVA revealed multivariate main effects of age group $F(5, 24) = 6.67$, $p < .0005$, cue condition, $F(10, 106), = 5.87$, $p < .0001$, and target type, $F(5, 24), = 9.60$, $p < .0001$. However, there was no multivariate age group by cue condition interaction $F(10, 106), = 1.58$, $p = .12$, no age group by target type interaction, $F (5, 24), = 1.09$, $p = .38$, and no cue condition by target type interaction, $F(10, 106), = 1.12$, $p = .35$. There was a significant multivariate three-way age group by cue condition by target type interaction, $F(10, 106), = 2.07$, $p < .05$. The significance of several effects in the multivariate

analysis allowed the inspection of the univariate mixed model Analysis of Variance (ANOVA) for each of the five dependent variables.

Prior to examining the results of each of the dependent variables it is important to note that the elderly subject group were administered only two experimental blocks of 60 trials per block while the young subject group were administered three blocks of 60 trials per block. This situation left open the possibility that any group effects may have been due to practice effects of the young subject group (i.e., they may have improved their performance by the third block). To investigate this possibility, a MANOVA was performed using only the data from the first two trial blocks of the young subject group and compared this to the elderly subject group's data (i.e., both age groups being compared on 2 trial blocks). The results did not differ from those using the complete data set for the young subjects. The results of the MANOVA using only 2 trial blocks of the young subject group with the elderly control group are as follows:

The multivariate MANOVA revealed multivariate main effects of age group $F(5, 24) = 6.58$, $p < .0005$, cue condition, $F(10, 106), = 6.06$, $p < .0001$, and target type, $F(5, 24), = 7.77$, $p < .0002$. There was no multivariate age group by cue condition interaction $F(10, 106), = 1.37$, $p = .21$, no multivariate age group by target type interaction, $F(5, 24), = 1.50$, $p = .22$, and no multivariate cue condition by

target type interaction, $F(10, 106)$, = 1.18, p = .31. There was a significant multivariate three-way age group by cue condition by target type interaction, $F(10, 106)$, = 2.03, p < .05. Given that these data were equivalent in pattern to those using all data from the young subject group, the univariate data presented below are the full set of data (i.e., 3 trial blocks for the young subject group and two trial blocks for the elderly subject group).

Reaction Time (See Figure 5.6)

There was a univariate main effect of age group for the RT measure, $F(1, 28)$ = 28.69, p < .0001. Comparison of the least squares adjusted means revealed that the young subject group's mean RT (M = 439ms, SE = 22.4ms) was 186ms faster, on average, than the elderly subject group's mean (M = 625ms, SE = 27.5; t = 5.25, p < .0001). There was no univariate age group by cue condition interaction, $F(2, 56)$ = .69, p = .46 and no age group by target type interaction, $F(1, 28)$ = 0.00, p = .95. An age group by cue condition by target type interaction was evident, $F(2, 57)$ = 6.48, p < .002. This interaction is partially explained by the fact that the pattern among the cue conditions differed between the two subject groups for both paradigms. For both subject groups, RT for the valid cue condition was faster than the invalid conditions for both target types. For the Exogenous Paradigm, the young subject group had a marginally slower RT for the invalid compared with the neutral cue condition (t = 9.75, p

=.08) while for the elderly subject group the invalid and neutral conditions did not differ (t = 9.75, p =.18). For the Endogenous Paradigm, for the young subject group, the invalid condition was faster than the neutral condition (t = 2.68, p <.01) while for the elderly subject group, the invalid condition was only marginally faster than the neutral condition (t = 1.89, p =.06). Also contributing to the interaction was the pattern of differences in the "costs plus benefits" between the two groups. The young subject group had larger "costs plus benefits" in the Endogenous Paradigm (74.3ms) than in the Exogenous Paradigm (55.3ms). A contrast comparing the "costs plus benefits" between the two target paradigms revealed that this difference approached statistical significance (F = 3.69, p = .07). This was reversed for the elderly subject group, who had greater "costs plus benefits" in the Exogenous Paradigm (101.5ms) than in the Endogenous Paradigm (63.4ms). Similar to the young subject group, a contrast of the "costs plus benefits" between the two paradigms for the elderly subject group revealed that this difference was marginally significant (F = 3.88, p = .06). Comparing the differences in the "costs plus benefits" for RT for the Exogenous Paradigm between the two groups revealed the differences to be marginally significant (F = 3.19, p = .08). For the Endogenous Paradigm, the differences in the "costs plus benefits" for the young and the elderly subject groups were not significant (F = 0.24, p = .63).

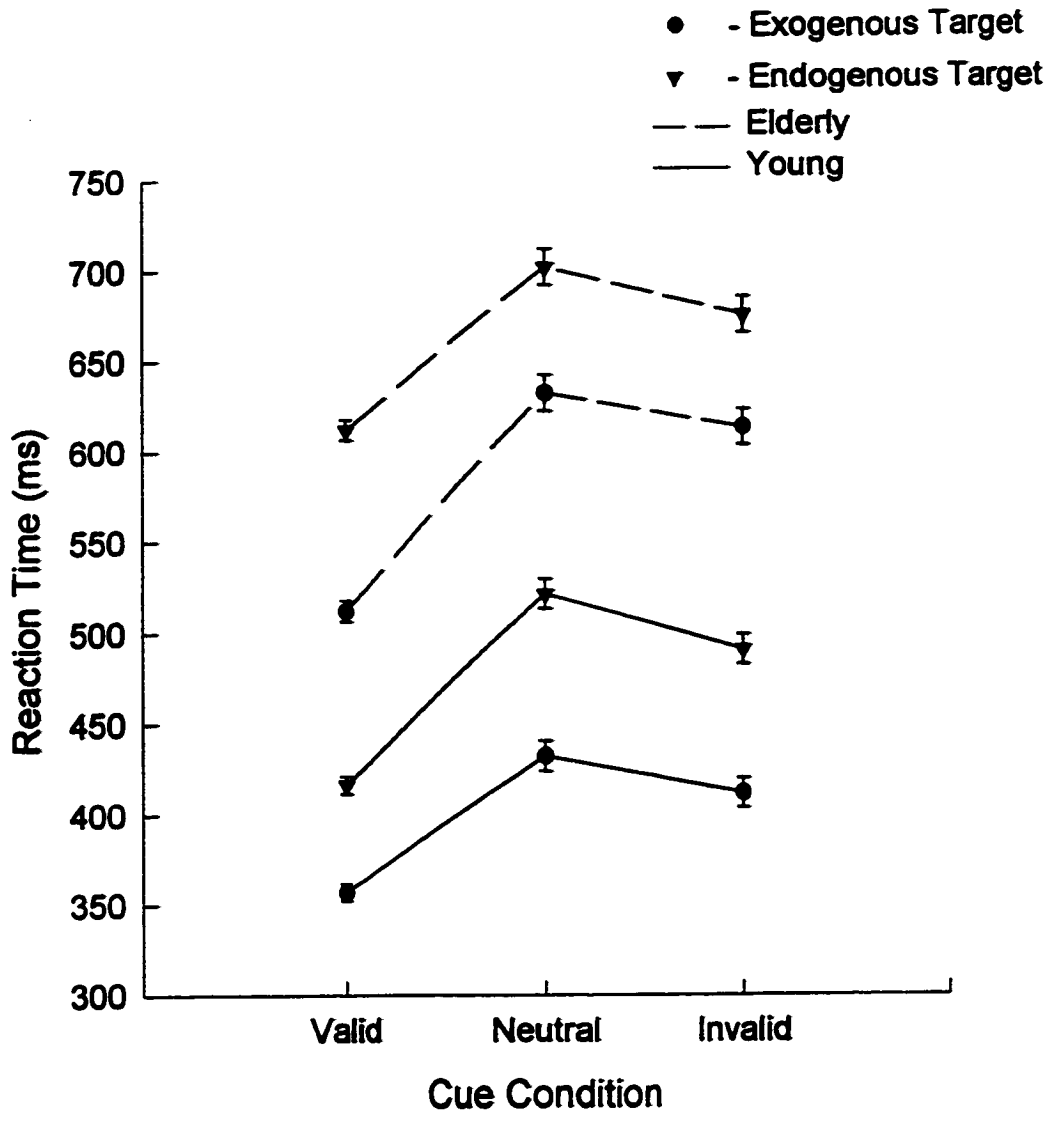


Figure 5.6 - Reaction time as a function of cue condition, target paradigm and age group. The error bars represent the standard error of the least squares means.

Movement Duration Time (See Figure 5.7)

There was a univariate main effect of age group for MT, $F(1, 28) = 18.15$, $p < .0002$. Paired comparisons using the least squares adjusted means indicated that overall MT was approximately 147ms shorter for the young subject group (M = 587ms, SE = 22.3ms) than for the elderly subject group (M = 734ms, SE = 27.3ms; $t = 4.17$, $p < .0005$) (collapsing across target type and condition). There was no age group by condition interaction, $F(2, 59) = 2.05$, $p = .14$, no age group by target type interaction, $F(1, 28) = .41$, $p = .53$, and no age group by cue condition by target type interaction $F(2, 59) = .95$, $p = .39$. This suggests that the pattern of performance was similar in the two groups. However, a priori planned paired comparisons revealed that the young subjects had significant "costs plus benefits" for both the Exogenous (12.3ms, $t = 2.88$, $p < .005$) and Endogenous Paradigms (26.7ms, $t = 6.97$, $p < .0001$) while the elderly subjects had no significant "costs plus benefits" for Exogenous Paradigm (3.8ms, $t = 0.75$, $p = .46$) and only a trend toward "costs plus benefits" for the Endogenous Paradigm (9.2ms, $t = 1.76$, $p = .08$). A statistical comparison of the differences in the "costs plus benefits" between the two groups for the Exogenous paradigm revealed the difference to be nonsignificant ($F = 0.95$, $p = .33$). For the Endogenous Paradigm, the difference in the "costs plus benefits" for the two groups was significant ($F = 5.45$, $p < .02$).

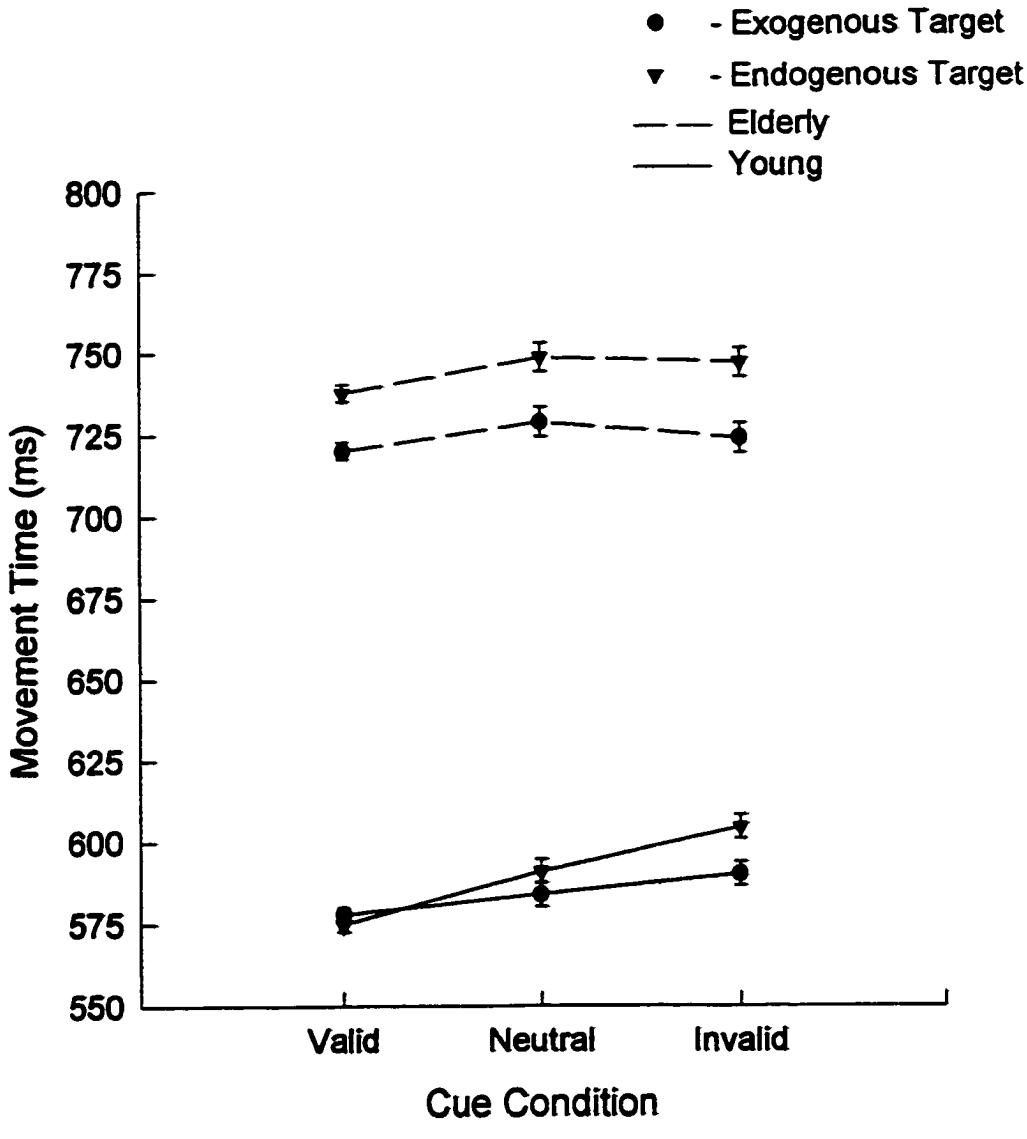


Figure 5.7 - Movement time as a function of cue condition, target paradigm and age group. The error bars represent the standard error of the least squares means.

Peak Velocity (See Figure 5.8)

There was a univariate main effect of age group for PV, $F(1, 28) = 10.85$, $p < .003$. A comparison of the least squared adjusted means revealed that PV was approximately 16.7cm/sec higher for the young subject group (M = 90cm/sec, SE = 3.3cm/sec) than for the elderly subject group (M = 73cm/sec, SE = 4.0cm/sec, $t = 3.22$, $p < .005$). There was also a univariate age group by cue condition interaction, $F(2, 59) = 5.80$, $p < .005$. This interaction is explained by the fact that for the young subject group, there were significant "costs plus benefits" in PV as a function of cue condition in the Exogenous Paradigm (1.3cm/sec, $t = 2.87$, $p < .005$) and the Endogenous Paradigm (3.0 cm/sec, $t = 6.69$, $p < .0001$) while for the elderly subject group, there were no differences as a function of cue condition in either the Exogenous (0.6cm/sec, $t = 1.15$, $p = .25$) or Endogenous paradigms (0.6cm/sec, $t = 1.08$, $p = .28$). Statistical comparisons of the differences in the "costs plus benefits" between the two groups for the Exogenous and Endogenous Paradigms revealed that they were significant ($F = 6.16$, $p < .02$ and $F = 7.95$, $p < .01$, respectively).

There was no age group by target type interaction, $F(1, 28) = 1.04$, $p = .32$ and no age group by target type by cue condition interaction, $F(2, 61) = .61$, $p = .55$.

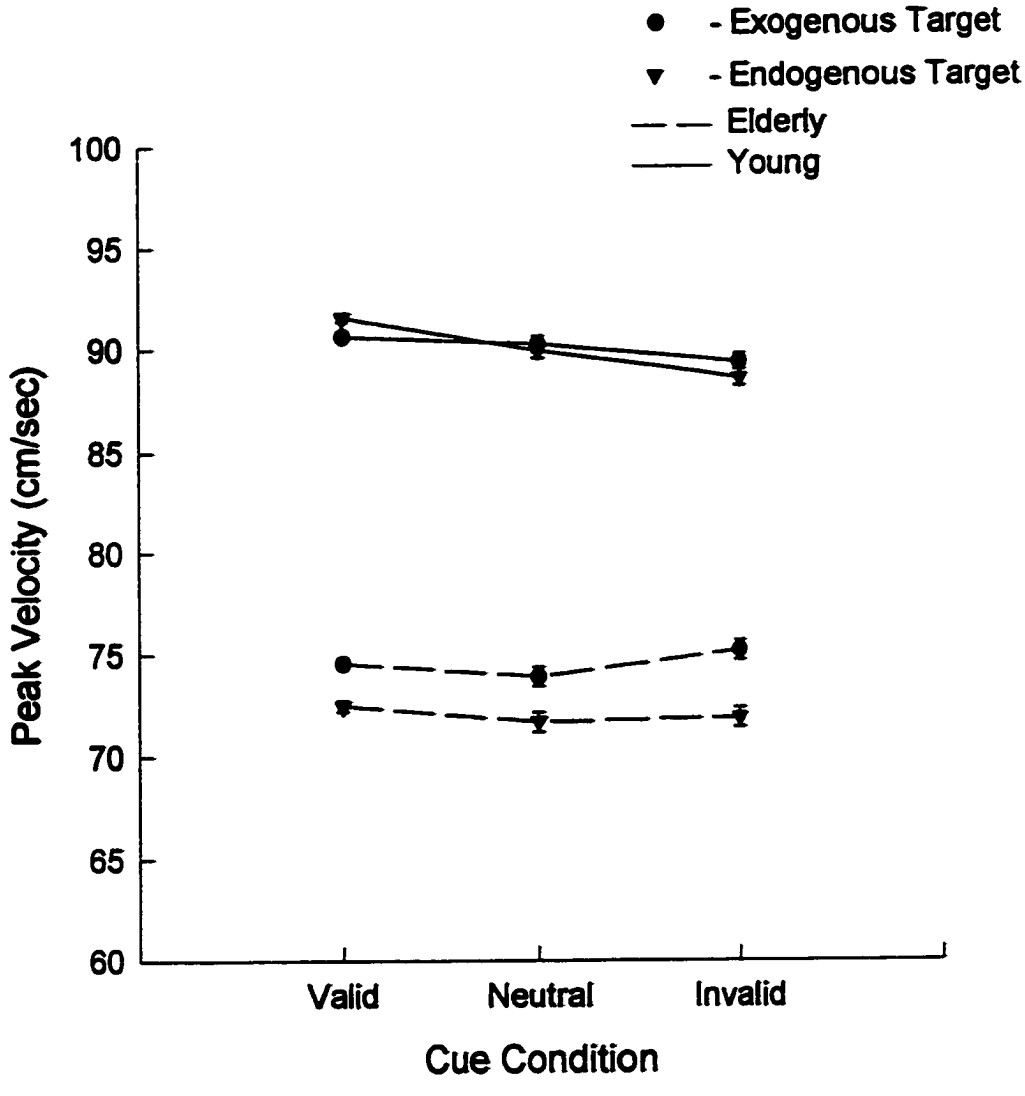


Figure 5.8 - Peak velocity as a function of cue condition, target paradigm and age group. The error bars represent the standard error of the least squares means.

Percent Deceleration (See Figure 5.9)

There was no univariate main effect of age group for percent deceleration, $F(1, 28) = 2.79$, $p = .11$, and no age group by cue condition interaction, $F(2, 58) = .81$, $p = .45$, no age group by target type interaction, $F(1, 28) = 2.59$, $p = .12$. The age group by cue condition by target type interaction was marginally significant, however, $F(2, 60) = 3.01$, $p = .06$. Paired comparisons using the least squares adjusted means revealed that the elderly subject group had significant "costs plus benefits" for percent deceleration in the Exogenous Paradigm (1.0%, $t = 2.53$, $p < .01$) while the young subject group did not (0.1%, $t = 0.40$, $p = .69$). For the Endogenous Paradigm, however, the elderly subject group had no "costs plus benefits (0.2%, $t = 0.61$, $p = .54$) while the young subject group did (1.6%, $t = 2.66$, $p < .01$). However, a comparison of the difference between the two groups for the "costs plus benefits" in the Exogenous and the Endogenous Paradigms revealed no significant differences ($F = 2.26$, $p = .14$ and $F = 0.98$, $p = .33$, respectively).

Resultant Error (See Figure 5.10)

There was no univariate main effect of age group for resultant error, $F(1, 28) = .58$, $p = .45$ and no significant interactions with age group (all p values $> .05$). Average error ranged from 7.4mm to 8.5mm among subjects and in both paradigms.

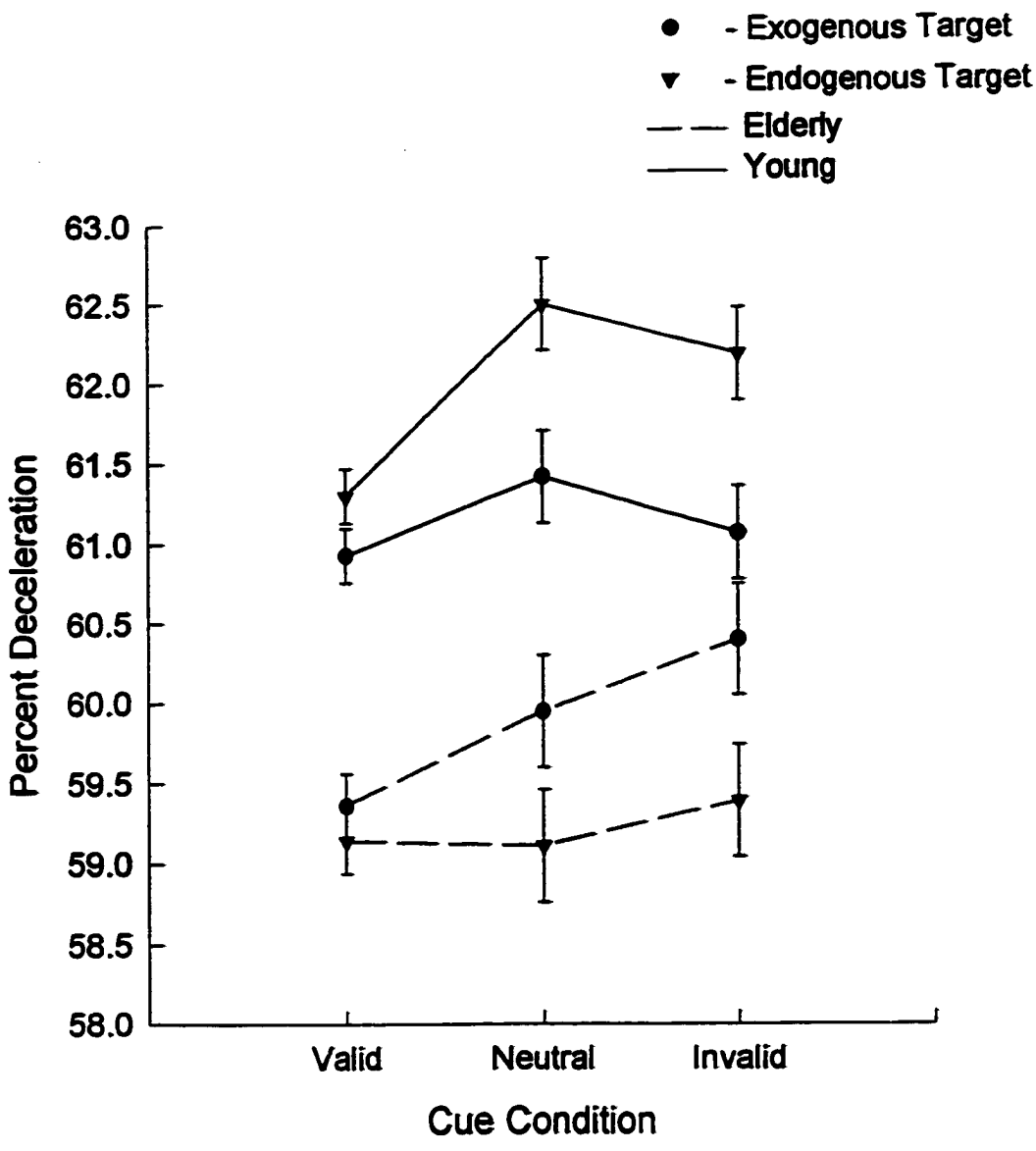


Figure 5.9 - Percent deceleration as a function of cue condition, target paradigm and age group. The error bars represent the standard error of the least squares means.

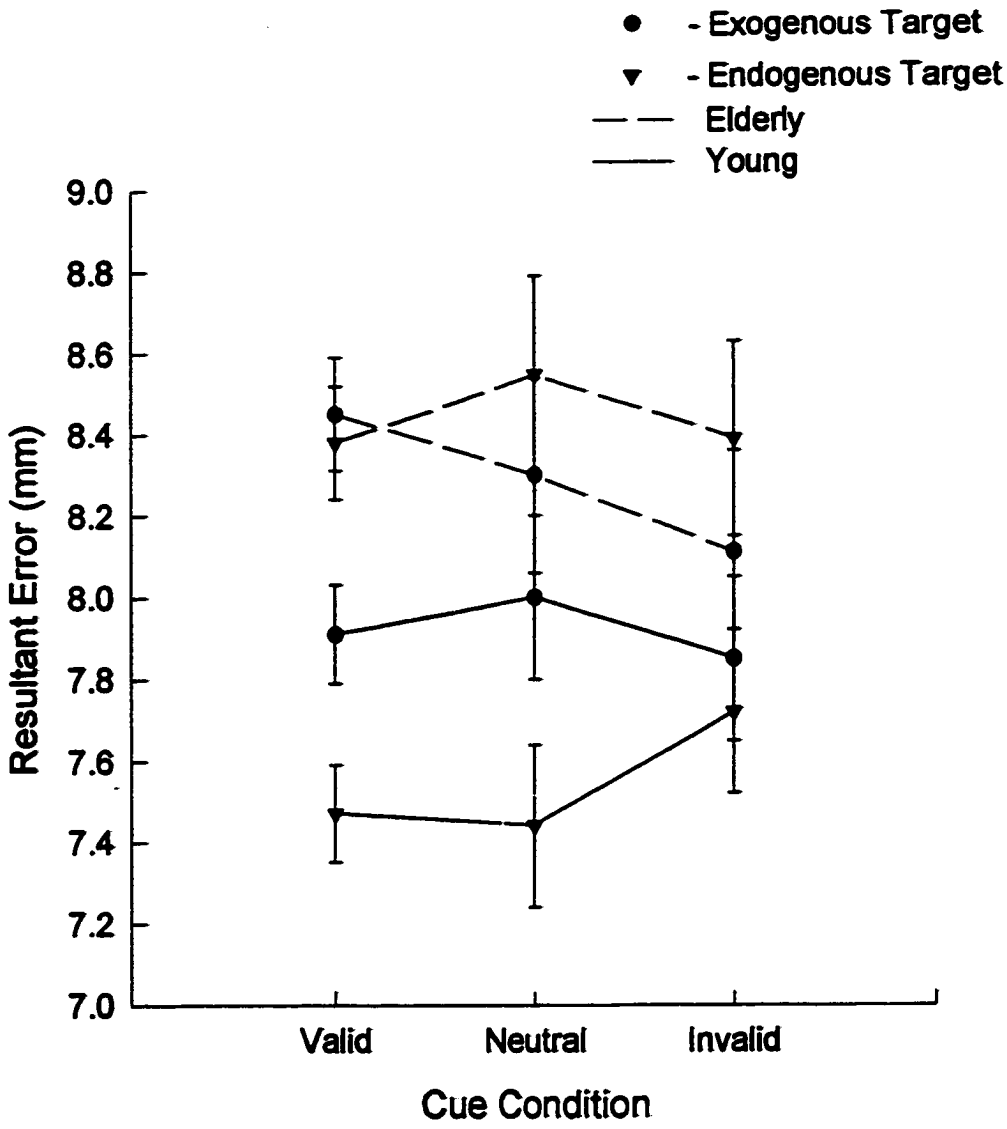


Figure 5.10 - Resultant error as a function of cue condition, target paradigm and age group. The error bars represent the standard error of the least squares means.

Total Performance Time (See Figure 5.11)

Given that it has been suggested that there are greater differences in RT between the young and the elderly than there are in MT (e.g., Warabi, Noda, & Kato, 1986), the following analysis of total performance time (RT plus MT) for the elderly and the young subject groups is of interest from an illustrative viewpoint. Figure 5.11 illustrates that the overall total performance time was approximately 33% greater for the elderly subject group (averaged across cue condition), and that most of this increase was due to the increased RT.

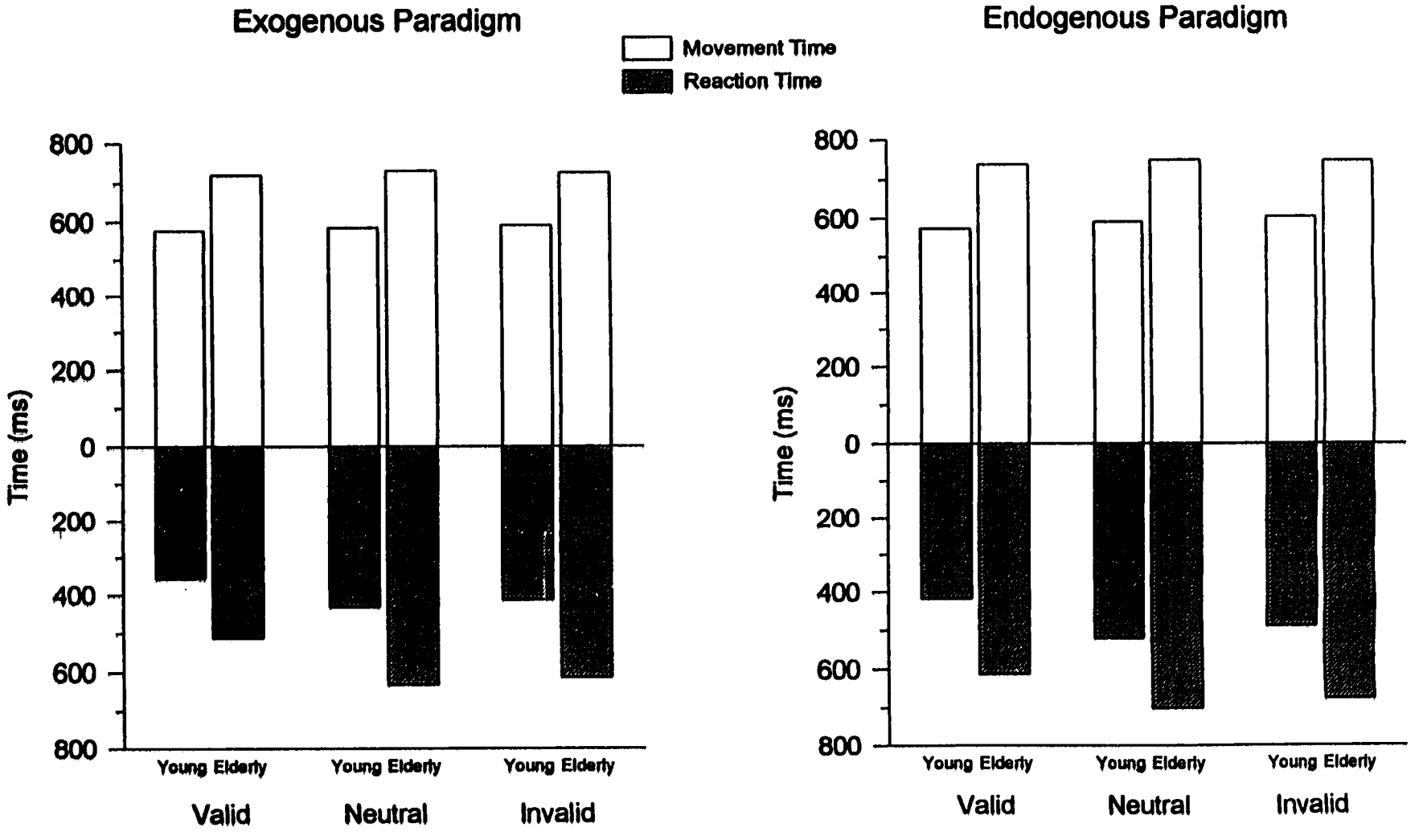


Figure 5.11 - Total Performance Time

ANALYSIS 3

HEMISPACE OF TARGET POSITION (See Table 5.1)

Analysis 3 was a 2 (Age Group - Young, Elderly) by 2 (Hemispace of Target Position - Left, Right) Multivariate Repeated Measures Analysis of Variance Design (MANOVA). Dependent measures were reaction time (RT), movement time (MT), peak velocity (PV), percent deceleration and resultant error. A series of planned paired comparisons using the least squares adjusted means was conducted to examine the differences between the hemispace of target position variables. Two variables were created for this analysis. "Target Left" included all trials for which the targets were 1 or 2. "Target Right" included all trials for which the targets were 3 or 4. These target variables were collapsed across cue type. See the previous chapter for the rationale for the creation of these variables.

The multivariate MANOVA indicated there to be a multivariate effect of age group, $F(6, 23) = 5.53$, $p < .001$, and of hemispace of target position $F(6, 23) = 32.09$, $p < .0001$ and a marginal age group by hemispace of target position interaction, $F(6, 23) = 2.16$, $p = .09$. There were no univariate interactions of age group and hemispace of target position for RT, $F(1, 28) = 1.65$, $p = 0.21$, MT, $F(1, 28) = 0.43$, $p = 0.51$, PV, $F(1, 28) = 0.26$, $p = 0.61$, percent deceleration, $F(1, 28) = 2.04$, $p = 0.16$, or resultant error, $F(1, 28) = 1.32$, $p = 0.26$. Post-hoc comparisons revealed that

percent deceleration was significantly longer for movements to targets in the ipsilateral hemispace for the young subject group (t = 3.91, p < .0005) while there was no difference in these variables for the elderly subject group (t = 1.39, p = 0.18).

Table 5.1

Hemispace of Target Position for Young and Elderly Groups

	RT (Ired #2)	RT (WATSCOPE)	MT	PV	%Decel	Error
Elderly Group						
Target Left	592.4[a]	729.6	758.3[b]	68.7[b]	58.7	8.4
SE	3.7	8.4	4.6	1.1	0.7	0.4
Target Right	607.3[a]	735.8	705.5[b]	73.0[b]	60.2	8.4
SE	3.7	8.5	4.7	1.1	0.7	0.4
Young Group						
Target Left	414.8[a]	450.5	606.4[b]	85.3[b]	59.8[a]	7.3[c]
SE	1.7	1.7	3.9	1.0	0.8	0.3
Target Right	421.2[a]	449.3	559.2[b]	95.6[b]	63.0[a]	8.1[c]
SE	1.7	1.7	3.9	1.0	0.8	0.3

[a], $p < .05$

[b], $p < .0001$

[c], $p = .06$

Same letter suprascript indicates significant differences.

ANALYSIS 4

MERIDIAN EFFECT

The meridian effect for the elderly subject group was examined in the same manner as was the young subject group. One comparison examined the meridian effect for contralateral reaches (i.e., target left of centre), and one examined the meridian effect for ipsilateral reaches (i.e., target right of centre). Each of these two comparisons included a contrast of the situation in which the cue and target were in the same hemispace (i.e., within-hemispace) with the situation in which the cue and the target were in the opposite hemispace (i.e., between hemispace). As with the young subject group, to investigate the meridian effect for contralateral targets, a within-hemispace cue and target condition (cue = 1, target = 2) was compared with a with a between-hemispace cue and target condition (cue = 3, target = 2). To investigate the meridian effect for ipsilateral targets, a within-hemispace cue and target condition (cue = 4, target = 3) was compared with a between-hemispace cue and target condition (cue = 2, target = 3).

Each analysis was a one-way (Meridian Type) Multivariate Repeated Measures Analysis of Variance (MANOVA). Dependent measures were RT, MT, PV, percent deceleration, and resultant error. A series of a priori paired comparisons using the least squares adjusted means was conducted to examine the differences between the "Meridians". Since only invalid trials

were used for this analysis the overall number of observations per cell was relatively small. Therefore this analysis has limited power and should be considered exploratory.

Results (See Table 5.3)

The multivariate effect for the within versus the between hemispace comparisons for contralateral targets for the Exogenous Paradigm was not significant $F(5, 10) = 1.24$, $p = 0.36$, nor was the multivariate effect for the within versus between hemispace comparisons for contralateral targets in the Endogenous Paradigm, $F(5, 10) = 0.62$, $p = .69$. The multivariate effect for within versus between hemispace comparisons for ipsilateral targets for the Exogenous Paradigm was not significant, $F(5,10) = 1.42$, $p = .30$. The multivariate effect for within and between hemispace comparisons for ipsilateral targets in the Endogenous Paradigm was marginally significant, $F(5, 10) = 2.73$, $p = .08$.

Examining the univariate effect for each dependent variable for the contralateral targets in the Exogenous Paradigm and the Endogenous Paradigm, revealed that none of the dependent measures were significant or even approached significance (all p values $> .05$).

Examining the univariate effect for each dependent variable for the ipsilateral targets in the Endogenous Paradigm, revealed that only PV was significant, $F(1, 14) = 5.00$, $p < .05$. As Table 5.2 illustrates, PV was higher for movements to targets where the cue and target were in the same

hemispace (79.2ms) compared with when they are in the opposite hemispace (75.7ms). All other dependent measures were nonsignificant (all p values > .05).

Table 5.2

Meridian Effect for Exogenous and Endogenous Targets as a Function of Hemispace of Target Position - Elderly Group

	RT	MT	PV	%Decel	Error
CONTRALATERAL TARGETS					
Exogenous					
Within	602.5	761.0	74.6	62.2	8.4
SE	35.2	14.4	0.8	0.9	0.6
Between	597.5	746.8	75.2	60.2	7.2
SE	34.3	13.9	0.8	0.9	0.6
Endogenous					
Within	650.2	757.8	72.3	58.3	8.3
SE	19.5	20.8	1.5	0.8	0.7
Between	674.5	778.1	70.9	56.9	8.8
SE	19.5	20.8	1.5	0.8	0.7
IPSILATERAL TARGETS					
Exogenous					
Within	586.7	707.9	81.6	60.7	8.3
SE	26.0	12.1	1.2	0.7	0.6
Between	538.0	696.5	82.6	59.3	7.1
SE	25.2	11.7	1.1	0.7	0.6
Endogenous					
Within	667.9	714.0	79.2[a]	60.5	7.2
SE	30.0	15.9	1.1	1.4	0.9
Between	717.0	728.2	75.7[a]	59.1	8.8
SE	30.0	15.9	1.1	1.4	0.9

[a] $p < .05$

ANALYSIS 5

A meridian analysis was also conducted on the combined elderly and young groups data. This analysis was a 2 (Meridian) X 2 (group) MANOVA (see Table 5.3).

The multivariate effect for the within versus the between hemifield comparisons for contralateral targets was not significant for the Exogenous Paradigm $F(5, 25) = 0.17$, $p = 0.97$ or the Endogenous Paradigm, $F(5, 25) = 0.36$, $p = .87$. Similarly, the multivariate effect for within versus between hemispace comparisons for ipsilateral targets was not significant for the Exogenous Paradigm, $F(5, 25) = 2.03$, $p = .11$ or the Endogenous Paradigm, $F(5, 25) = 1.48$, $p = .23$. Examining the univariate effect for each dependent variable for the contralateral targets in the Exogenous Paradigm and the Endogenous Paradigm, revealed that none of the dependent measures were significant or even approached significance (all p values $> .05$).

Examining the univariate effect for each dependent variable for the ipsilateral targets in the Exogenous Paradigm type, revealed that MT was significant, $F(1, 31) = 4.41$, $p < .05$ and PV was marginally significant, $F(1, 31) = 3.23$, $p = .08$. As Table 5.3 illustrates, PV was higher for movements to targets where the cue and target were in opposite hemispaces (90.2ms) compared with when they were in the same hemispace (88.3ms). All other dependent measures were nonsignificant (all p values $> .05$).

Table 5.3

Meridian Effect for Exogenous and Endogenous Targets as a Function of Hemispace of Target - Elderly and Young Groups

	RT	MT	PV	%Decel	Error
CONTRALATERAL TARGETS					
Exogenous					
Within	498.9	682.8	81.9	60.3	7.9
SE	16.7	8.1	0.6	0.6	0.5
Between	503.4	677.3	81.8	60.0	7.6
SE	16.3	7.9	0.5	0.6	0.5
Endogenous					
Within	577.4	691.1	80.6	59.9	7.9
SE	10.4	11.0	1.1	0.8	0.4
Between	594.3	699.2	80.1	59.9	7.4
SE	10.4	10.9	1.2	0.7	0.4
IPSILATERAL TARGETS					
Exogenous					
Within	493.9	651.2[a]	88.3[b]	61.2	7.9
SE	13.1	8.3	0.8	0.5	0.3
Between	471.5	626.8[a]	90.2[b]	60.3	7.3
SE	13.0	8.2	0.7	0.5	0.3
Endogenous					
Within	572.9	649.7	86.5[a]	61.4	7.7
SE	15.2	8.6	0.8	0.7	0.5
Between	596.1	665.7	84.1[a]	60.6	8.4
SE	15.1	8.6	0.8	0.8	0.5

[a] indicates a trend toward significance, (p = .05).

[b] indicates a trend toward significance, (p = .08).

DISCUSSION

The healthy elderly group had overall faster RT for the Exogenous compared with the Endogenous Paradigm. They had larger "costs plus benefits" for the Exogenous compared with the Endogenous Paradigm. This indicates that they did shift attention in response to the cues in the Exogenous Paradigm. As well, this suggests that they received an added advantage of having shifted attention plus programmed a response prior to movement initiation (i.e., Alternative #1, pg. 48) compared with programming a motor response only (i.e., Alternative #1, pg. 51).

No statistically significant differences in the pattern of performance as a function of hemispace of target presentation were noted between the young and the elderly adults used in this study.

There was no meridian effect for the elderly subject group. It must be noted that the power of this analysis was low to detect any effects. When combined with the young group's data, the pattern was identical to the young group.

Main effects indicated that the elderly subject group were, overall, slower to initiate movements (by approximately 186ms), took longer to complete movements (by approximately 147ms), and reached a lower average peak velocity (by approximately 17cm/sec) when compared to the young subject group. All of these differences are comparable to those noted by other researchers for RT (e.g., Amrhein et al., 1991, 1993;

Folk & Hoyer, 1992; Stelmach et al., 1988), MT (Amrhein et al., 1991; Roy et al., 1993) and PV (Roy et al., 1993). These differences associated with aging have been attributed to age-related neural and muscular changes (e.g., neuron and synaptic loss; decreased number and size of muscle fibres) resulting in slower information processing speed (e.g., Roy et al., 1993), a cautious approach (e.g., Larish & Stelmach, 1982; Walker et al., 1997) and a deficit in the ability to control the generation of force (Pratt et al., 1994; Roy et al., 1993; Stelmach et al., 1988; Walker et al., 1997) compared with the young.

That there were no differences in the deceleration phase between the elderly and the young in this study is in contrast to many studies that have noted a longer percent deceleration for elderly subjects (e.g., Bennett & Castiello, 1994; Brown, 1996; Roy et al., 1993; Warabi et al., 1986). The equivalent deceleration phases for the young and the elderly subject groups in this study suggest that the elderly subject group did not require more time to process feedback than the young subject group as was suggested by Roy et al. (1993), or to make more end-point corrections to their movements in order to maintain equivalent accuracy to the young. That there were no differences in accuracy between the elderly and the young subject groups is in contrast to studies that have noted either greater accuracy in elderly subjects (Bennett & Castiello, 1994; Tellinghuisen et al., 1996) or greater errors

for elderly subjects (e.g., Goggin & Stelmach, 1990; Stelmach et al., 1987, 1988). The equivalent accuracy between the two groups in this study is consistent with results of Amrhein and Goggin (1988) and Smith and Brewer (1995) and is also consistent with the equivalent deceleration times noted (since greater accuracy is often associated with longer deceleration time). Furthermore, equivalent accuracy for the elderly and the young groups suggests that the elderly group was not more concerned with accuracy than were the young group.

Longer MT for elderly subjects (compared with young subjects) has been attributed to deficits in processing feedback (Larish & Stelmach, 1982; Stelmach et al., 1987) and to deficits in the ability to generate force (Pratt et al., 1994; Goggin & Stelmach, 1990; Stelmach, Goggin & Amrhein, 1988). Since a deficit in the ability to process feedback has also been associated with longer deceleration phases (which were not evident in the current study), the longer MT for the elderly, in this study, seems most likely attributable to a deficit or reduction in force production. This suggestion is supported by the lower average PV of the elderly subjects in this study.

That a greater proportion of the increase in total performance time was accounted for by increased RT (compared with MT) is consistent with the findings of Warabi et al., (1986) that there are greater differences in RT between elderly and young adults compared with MT. These

proportionately greater increases in RT for the elderly may be an indication that they were more deliberate in identifying the response required, prior to initiating the movement (Larish & Stelmach, 1982), required a longer time to process the cued information, or had to interpret the cue and program the movement in a sequential manner (within the RT interval). In contrast, young subjects may have been able to interpret the cue and prepare the response simultaneously (for review see Spirduso & MacRae, 1990). This pattern may also indicate that the physiological changes associated with aging increase the time needed for the processes which take place during the RT interval including: identifying the response required, choosing the appropriate response, transmitting the response to the muscles, and activating the muscles; (Welford, 1988) or to difficulty generating force at movement initiation (Roy et al., 1993).

It is likely that the results of this study reflect a combination of the above processes rather than to any one process specifically. In summary, the results for the elderly in the current study appear consistent with the idea that the elderly have longer MT as a result of a reduced ability to generate force, and have longer RT due to a combination of a cautious or deliberate approach, slowing of the processes that take place prior to initiating a movement (Welford, 1988) and/or interpreting the cued information, and to reduced force generation at movement initiation.

Cuing Effects

Results of the current study indicated that the elderly subjects were able to use cues to facilitate both exogenous and endogenous overt movements. This indicates that they were able to covertly shift visual attention in response to endogenous visual cues and to preprogram movements using pre-movement information. This is consistent with other researchers who have noted similar results for studies of shifts of visual attention (e.g., Folk & Hoyer, 1992, experiment 3; Nissen & Corkin, 1985; Robin & Rizzo, 1992) and for studies of movement preparation precuing (e.g., Gottsdanker 1980a; Larish & Stelmach, 1982; Stelmach et al., 1987).

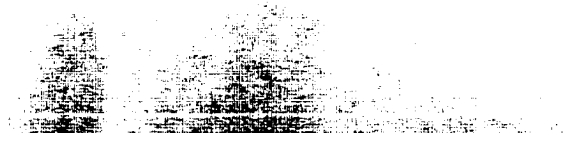
The advantage of having been provided with valid cues (for both exogenous and endogenous targets) for the elderly subjects in this study, was confined to the RT interval. This suggests that the elderly subjects completed all reprogramming of an incorrectly preplanned movement during the RT interval, consistent with the suggestion of Larish and Stelmach (1992). This result was in contrast to the young subjects who appeared to complete only some of the reprogramming prior to movement initiation but made further adjustments during movement execution. As discussed previously, this may have been due to strategy differences between the two groups. It is also possible that the elderly subjects in this study, when cued invalidly, aborted the invalid program and made a new program

from "scratch" when the target was presented. The fact that RT was equivalent for both the neutral and the invalid conditions in elderly subjects would lend support to this argument (as there was, presumably, no pre-programming completed in RT interval of the neutral condition). However, as discussed previously, one cannot make firm conclusions based on any of the comparisons with the "neutral" cues used in this study.

Exogenous Versus Endogenous Movements

As with the young subjects, the elderly subjects had faster RT to the exogenous compared with the endogenous targets (collapsing across cue). This indicates that the advantage of abrupt onset targets that have been noted for young adults (e.g., Jonides & Yantis, 1988; Juola et al., 1995) remains intact with healthy aging.

Examining the magnitude of the RT "costs plus benefits" (i.e., invalid RT minus valid RT) for the two target types revealed different patterns for the young and the elderly subject groups. For the elderly subject group, there was a trend towards the RT "costs plus benefits" being greater for the Exogenous Paradigm (101.5ms) than the Endogenous Paradigm (63.4ms). For the young subject group, this pattern was reversed with there being a trend towards the "costs plus benefits" for the Exogenous Paradigm (55.3ms) being less than for the Endogenous Paradigm (74.3ms). This suggests the possibility that the young subject group may not have covertly shifted their attention in response to the cues while the



elderly control group did. However, another alternative interpretation for the performance of the young subject group, as discussed in the previous chapter is that the young subject's "costs plus benefits" for the Exogenous Paradigm reflected only a shift of attention with little, if any, programming while the opposite was true of the Endogenous Paradigm. In contrast, the elderly subjects may have specified the motor programming similarly in both paradigms but shifted attention in the Exogenous Paradign only. The difference between the "costs plus benefits" for the Exogenous and Endogenous Paradigms for the elderly control group was 38ms which could be considered the added effect of a shift of attention in addition to motor programming only. Given that the RT "costs plus benefits" were of similar magnitude for the young and elderly groups for the Endogenous Paradigm suggests that these two groups did not differ with regards to motor programming/reprogramming prior to movement initiation (since the Endogenous Paradigm involved only motor programming). Unfortunately, it was not possible to isolate the effects of separate components of shifts of attention (engagement, shift, or disengagement) in this study because the neutral cues did not provide an appropriate baseline measure.

The similar patterns of differences in "costs plus benefits" between young and elderly subjects that were revealed in the current study for the Exogenous Paradigm (i.e., greater "costs plus benefits" for elderly subjects

compared with young subjects), have also been noted by other researchers (Brodeur & Enns, 1997; Folk & Hoyer, 1992; Hartley et al., 1990). However, the attentional components considered responsible for these findings have differed. For example, Brodeur and Enns (1997) who used exogenous cues paired with exogenous targets and endogenous cues paired with exogenous targets concluded that the "orienting effect" was larger for the elderly than the young for exogenous cues and larger for young than elderly subjects for endogenous cues. They did not relate their results to specific mechanisms of attentional orienting. Folk and Hoyer (1992), experiment 3, pairing endogenous cues with exogenous targets, noted that the "costs plus benefits" were almost double for elderly subjects compared with young subjects and interpreted this to indicate that "older adults may be more sensitive to spatial cuing than young adults" (p. 462) in that they were slower to process the cues and were influenced by the complexity of the cue. Hartley et al. (1990), pairing endogenous cues with exogenous targets (experiment 2), also noted larger "costs plus benefits" for elderly subjects compared with young subjects. They discussed their results in terms of attentional resource models, not of components of attentional processes per se. They interpreted their results to indicate that elderly adults shift more attentional resources as a function of cues than do young adults.

CONCLUSIONS

Results of the hemispace of target position analysis were similar for the elderly and the young subject groups (i.e., no interactions), suggesting that the integrity of this system remains intact with healthy aging. The advantage of abrupt onset targets that was revealed for the young subject group was also noted for the elderly subject group, suggesting that this advantage is resistant to aging. The elderly and young subject groups used in the current study had constant accuracy across the cue conditions and the two target paradigms. As discussed in Chapter 4 (young subject group), this suggests that constant accuracy was a goal of movements for both young and elderly subjects.

The elderly subject group had longer RT, slower MT, and lower PV than the young subject group. These findings are consistent with those noted by previous researchers and with the cognitive and physiological changes that have been attributed to aging (e.g., decreased size and number of muscle fibres, decreased neurons, fewer neuronal interconnections, slower processing speed).

Results suggest that the elderly subject group completed all reprogramming of both exogenous and endogenous overt movements prior to initiating movements. This is in contrast to the young subject group who appeared to complete some reprogramming prior to movement initiation but made further adjustments during movement execution. This may have resulted

from the elderly using a more cautious or deliberate strategy than the young or to the physical changes associated with the aging process or to a combination of both of these factors.

The elderly subject group showed a particular advantage (for the valid cue condition) for the initiation of movements when shifting attention in addition to motor programming (i.e., Exogenous Paradigm) in comparison to motor programming only (i.e., Endogenous Paradigm). That is to say that, for the elderly control group, there was an advantage of having prepared a motor response in the valid condition of the Endogenous Paradigm, but that there was an added advantage of having shifted attention and programmed a response in the valid condition of the Exogenous Paradigm.

Understanding the age-related changes in cognitive-motor behavior (such as those noted in the current study) is particularly relevant to the study of neurodegenerative processes that affect higher level cognitive functioning (e.g., Alzheimer's disease) as well as those that affect motor functioning (e.g., Parkinson's disease).

In the following two chapters, measures of covert shifts of visual attention and motor programming and reprogramming in healthy elderly adults will be compared with individuals with Alzheimer's Disease and Parkinson's Disease.

Chapter 6 - Study 3

THE EFFECTS OF VISUAL ATTENTION AND MOTOR PROGRAMMING ON MANUAL AIMING MOVEMENTS IN INDIVIDUALS WITH ALZHEIMER'S DISEASE

INTRODUCTION

Alzheimer's Disease (AD) is a progressive neuro-degenerative disease of gradual onset for which there is no cure. It was named for Alios Alzheimer who, in 1907, first described the relationship between neurofibrillary tangles and symptoms of dementia in a 55 year old woman. Estimated prevalence rates of AD in Canada are 5% (50/1000) of those over the age of 65 (Canadian Study of Health and Aging Working Group, 1994a). AD makes up 64% of all cases of dementia (Canadian Study of Health and Aging Working Group, 1994a). AD limits life expectancy (Corkin, 1981) and has been reported to be the fourth leading cause of death among the elderly in the United States (Richards & Stern, 1992). There is no known cause of AD but hypothesized etiologies include both environmental exposure to various agents (e.g., Bayles, Kaszniak, & Tomoeda, 1987; Canadian Study of Health and Aging, 1994b), and genetic susceptibility or heritability (Seshadri, Drachman & Lippa, 1995).

Diagnosis of AD

Various diagnostic criteria for dementia have been used but invariably dementia is characterized by the existence of multiple cognitive deficits. According to the most widely used

criteria (Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, 1994 - DSM-IV), a diagnosis of dementia of the Alzheimer's type requires the presence of a memory impairment plus one of the following: apraxia (an inability to perform purposive movements in the absence of motor or sensory impairments), aphasia (impairment in the ability to communicate through speech), agnosia (loss of comprehension of auditory, visual, or other sensations although the sensory sphere is intact), or a deficit in executive functioning. The deficits must be severe enough to cause an impairment in occupational or social functioning, and must represent a decline from the previous level of cognitive functioning. Those with dementia may have also have difficulty with spatial tasks and temporal orientation although these are not included in the DSM-IV criteria (APA, 1994). However, impaired spatial (i.e., constructional) ability was one of the criteria for diagnosis of AD in the DSM-III-R (APA, 1987).

Although a diagnosis of definite AD is possible only from autopsy (evident from the existence of numerous plaques and tangles in the neocortex and hippocampus) even pathological criteria can vary (e.g., Crystal, Dickson, Fuld, Masur, Scott, Mehler, Masdeu, Kawas, Aronson, & Wolfson, 1988; Molsa, Sako, Palijarvi, Rinne & Rinne, 1987). Nevertheless, reasonably valid diagnostic criteria for clinically probable and possible AD have been established by the National Institute of Neurological and Communicative Disorders and Stroke, and by

the Alzheimer Disease and Related Disorders Association (McKhann, Drachman, Folstein, Katzman, Price, & Stadlan, 1984). These criteria are as follows: For probable AD, the presence of dementia must have been established by clinical examination, documented by a mental status questionnaire, and confirmed by neuropsychological tests; the presence of deficits in two or more areas of cognition; progressive worsening of memory and other cognitive functions; no disturbance of consciousness; onset between ages 40 and 90 but most often after the age of 65; and, an absence of systemic disorders or other brain diseases that could account for the progressive deficits in memory and cognition. The criteria for possible AD include: the presence of dementia in the absence of neurologic, psychiatric, or systemic disorders that could cause the dementia and in the presence of variations in onset, presentation and clinical course; or, the presence of dementia when there is a systemic or brain disorder which is not considered to be the cause of the dementia. Both Blacker and colleagues (Blacker, Albert, Bassett, Go, Harrell, & Folstein, 1994) and Schofield and colleagues (Schofield, Tang, Marder, Bell, Dooneief, Lantigua, Wilder, Gurland, Stern, & Mayeux, 1995) found that clinical diagnoses based on these criteria were confirmed by autopsy in over 90% of the cases.

Treatments for AD

While there is no cure for AD, there are numerous pharmacological treatments under development that focus on

symptom relief. Many of these drugs attempt to increase the level of available acetylcholine in the CNS. An example of this is ARICEPT™ which represents the first approved treatment for the symptoms of AD in Canada. Other drugs under development are neurotrophins that are thought to promote the survival and the growth of neurons and may thereby delay or halt the progression of AD (Ainsen & Davis, 1997; see Feldman, Meyer, & Quenzer 1997 for a review). Recent work has also suggested a possible role for anti-inflammatory medications in slowing the process of AD (see McGeer & McGeer, 1997 for a review).

Neurophysiology and Neurochemistry of AD

Neurophysiologically, AD is characterized by the existence of bilaterally distributed neuritic plaques (groups of degenerating neurons surrounding an amyloid core), neuro-fibrillary tangles (bundles of paired helically-wound filaments within a cell body) and areas of granulovacular degeneration (fluid-filled spaces and granular debris within the cell, most often in the hippocampus) (Bayles et al., 1987). Neuro-fibrillary tangles are considered to be the hallmark of AD (Iqbal & Wisniewski, 1983) and are found in the pyramidal cells of the hippocampus and the amygdala. Neuritic plaques are found primarily in the outer half of the cortex (especially in the 3rd layer of the frontotemporal cortex) (Bayles et al., 1987), the hippocampus (Wisniewski, 1983), and the pulvinar (Kuljis, 1994). The degenerative neuronal process

involves primarily axonal terminals or pre-terminals (Bayles et al., 1987). There is a loss of large neurons primarily in the frontal and temporal cortical areas (especially the association areas) and a reduction in the number of dendritic branches. Neuroradiological investigations may reveal reduction of total brain volume with a dilation of the lateral ventricles, shrunken gyri, and widened sulci.

Although AD is typically thought of as a neuro-degenerative process of the cortex, there is also neuronal loss in subcortical areas, including the basal forebrain, the locus coeruleus, the raphi nuclei (dorsal raphe) and parts of the hypothalamus (Katzman, 1986a). In the entorhinal cortex, the large pyramidal neurons in layer II that project to the hippocampus degenerate. In the basal nucleus there is a degeneration of the larger cholinergic cells that project to the cerebral cortex.

The neuroanatomical changes in AD are accompanied by neurochemical changes. Postmortem studies have indicated that there is a reduction (50%-90% of that of normal controls) in choline acetyltransferease (the enzyme that produces and releases acetylcholine) in the cortex and the hippocampus (Katzman, 1986a), and a reduction of cholinergic neurons in the nucleus basalis of Meynert. This reduction has been strongly linked with the severity of dementia (see Whitehouse, Price, Clark, Coyle, & Delong, 1991 for a review), which is consistent with the evidence linking the cholinergic system to

memory and learning processes (Sims & Bowen 1983). Such findings have provided the rationale for cholinergic replacement as a potential pharmacotherapy for AD. The cholinergic innervations of the cortex arise from the medial basal forebrain (medial septal nucleus, nucleus of the diagonal band of Broca, nucleus basalis of Meynert) which is composed of a sheet of cholinergic neurons that innervate the hippocampus, amygdala, and neocortical areas (Katzman, 1986a). AD has been associated with a deficit in the function of the presynaptic rather than postsynaptic cholinergic neurons (Bayles et al., 1987).

Although most research has focused on the cholinergic deficits in AD, it has been suggested that there is also a reduction in noradrenaline and serotonin in the frontal and temporal cortex. A reduction in dopamine-B-hydroxylase (the marker enzyme for the noradrenergic transmission) has been noted, although this reduction is less than that of choline acetyl transferase. Noradrenergic cells are found in the nucleus coreuleus and function to influence learning, memory and attention (Bayles et al., 1987; Lawrence & Sahakian, 1995).

Behavioral and Cognitive Changes in AD

Behavioral changes in AD often begin subtly. The first sign is often memory loss for recent events, although, on rare occasions, deficits in language or constructional abilities may be evident first (Corkin, 1981). With disease progression,

the ability to learn new information and to recall past events and knowledge deteriorates. Orientation for time and place becomes impaired, as do judgement, reasoning, and abstract thought processes. These changes result in impaired performance of complex and novel tasks (Hart & Semple, 1990). Language functions, visuospatial perception and, praxis (the ability to plan and execute coordinated movement) also deteriorate (Katzman, 1986b), until, eventually, all cognitive functions become impaired (Hart & Semple, 1990).

Motor Programming and Apraxia in AD

It has been claimed that individuals in the early stages of AD have intact motor output systems, on the basis of measures of finger tapping speed, foot tapping speed, grip strength, and hand and head steadiness (Hom, 1992; Kluger, Gianutsos, Golomb, Ferris, George, Franssen, & Reisberg, 1997). However, early stage AD patients have also been shown to be impaired relative to controls on fine motor tasks and more complex motor tasks that require attentional, visuo-spatial and motor control mechanisms (e.g., grooved pegboard, choice reaction time, rotary pursuit, Purdue pegboard, alternating hand movements, head tracking) (Kluger et al., 1997; Pirozzolo, Mahurin, & Swihart, 1991 for a review). Kluger at al. (1997), using discriminant analysis, found that complex motor tasks were as efficient as tests of cognitive ability in discriminating mild AD patients from controls, indicating that the performance of psychomotor tasks declines

early in AD. As well, Kluger et al., (1997) found no correlation between prior level of education and performance on motor tests, providing an advantage over some other cognitive tests which do correlate with prior education level. Such correlations have contributed to the controversy surrounding epidemiologic studies which show a lower level of education is associated with an increased risk of AD (e.g., The Canadian Study of Health and Aging, 1994b).

The posterior parietal cortex is thought to be involved in visuomotor integration including motor planning and coordinate transformation (Andersen, 1993). This area is known to be affected in those with mild AD (Haxby, Grady, Koss, Horowitz, Schapiro, Friedland, & Rapoport, 1988). Thus, it is not surprising that mildly affected AD patients have difficulty on numerous motor tasks.

The deficit in motor functioning most typically revealed in clinical neuropsychological assessments of patients with AD is manual apraxia. Manual apraxia refers to the inability to conduct movements (either meaningful or not) which is not attributable to physical weakness, mobility problems, inattention to commands, or poor language comprehension (Kimura, 1993). It is often assessed by having people pantomime everyday simple activities such as waving, slicing bread, or brushing ones teeth.

Two types of apraxia are commonly described. Ideomotor apraxia, the most commonly referred-to form of apraxia, refers

to the inability to generate a correct movement, either to verbal command or by copying from demonstration. In contrast, ideational apraxia refers to an impairment in the overall plan of a complex series of movements. Kimura (1993), however, suggests that ideational apraxia is, in fact, merely a milder form of ideomotor apraxia which only becomes evident in the most complex circumstances.

Apraxia is considered to be a deficit in movement selection rather than a deficit in movement execution. The neural systems involved in praxis have been considered to be located primarily in the left hemisphere because of the common association of apraxia and aphasia (which is localized to the left hemisphere). Support for this assertion comes from the fact that although apraxia is associated with damage to the left hemisphere, it is usually expressed bilaterally in the limbs.

While apraxia is considered a clinical sign of dementia and AD, it has often been poorly operationalized or overlooked in studies of AD (Tuokko, Kristjamsson, & Miller, 1995), and has received relatively little attention in the body of research on cognitive deficits of AD (Benke, 1993; Foster, Chase, Patronas, Gillespie, & Fedio, 1986) perhaps because ideomotor manual apraxia has generally been considered to occur in the later stages of AD (for a review see Zec, 1993).

Kinematic Studies of Apraxia

Kinematic analyses have detected subtle differences

between patients with different types of neurologic damage that were not evident on traditional neuropsychological tests (Goodale, Milner, Jakobson, & Carey, 1990). Morever, kinematic studies and have illustrated subtle differences in movement programming and execution among such clinical groups (Fisk & Goodale, 1988; Goodale et al., 1990). This suggests that kinematic analyses have the potential to be particularly valuable for testing clinical groups (such as the AD and PD groups included in this study).

Heilman and colleagues (Poizner, Mack, Verfaellie, Rothi, & Heilman, 1990) were the first to investigate apraxia using kinematic analysis techniques similar to those of the present study, although not with demented patients. They had two subjects with left hemisphere lesions pantomime movements involving tool use to verbal command (e.g., unlocking a door with a key). Their results showed that these persons with apraxia had deficits in: spatial orientation (i.e., they oriented the tool inappropriately with their body), in distal joint control (i.e., they used proximal muscles to control distal muscles), in movement initiation (i.e., initial hesitation), and in spatiotemporal relationships (inappropriate velocity for the degree of required curvature of the movement trajectory). As well, they found that apraxic subjects had greater deficits in movements using distal, compared with proximal, musculature.

To date, only one study has examined the kinematics of

movement in a complex response planning task for patients with AD. Bellgrove, Phillips, Bradshaw, Hall, Presnell, and Hecht (1997) conducted a kinematic analysis of response programming in 12 subjects with a diagnosis of probable AD and 12 controls. AD subjects were screened for cognitive status using the Mini Mental Status Examination (MMSE) (Folstein, Folstein, & McHugh, 1975) (scores ranged from 14 to 27). Subjects were required to connect a series of 4 target light emitting diodes (LED's) with a pen on a graphics tablet. They were instructed to move quickly and accurately and to aim for the centre of the target. To examine the effect of precision, large and small targets were compared. In the "cued" condition, a series of four LED's was illuminated, one at a time, following the subjects movement to each successive target. In the "no cue" condition, one LED was always illuminated as the start position. In order to illuminate the next LED in the series, subjects were required to move their hand away from the current target. Bellgrove et al. (1997) reasoned that, in the cued condition, subjects would begin to program the movement to each subsequent target prior to starting their movement while in the no cue condition, they would begin the movement but had to reprogram it during execution once the target was illuminated. A measure of "pause time" (i.e., time spent at one target prior to beginning to move to the next) was thus thought to indicate the time needed to disengage attention from the previous target, identify the next target, and

prepare the movement. The total "movement time" (i.e., time spent moving between targets, excluding all pauses) was considered to be a measure of movement execution time during which reprogramming was thought to take place. They constructed a measure which they termed the "force inefficiency index" (i.e., the ratio of the number of zero crossings in the acceleration profile to the number of zero crossing in the velocity profile) and proposed this as an index of the number of submovements made. They also analyzed a measure which they termed the "asymmetry ratio" (i.e., time to peak velocity divided by movement time) which indicated the proportion of the movement conducted during the acceleration phase. Accuracy was also measured.

Bellgrove et al.'s (1997) results indicated that "pause times" were shorter in the cue conditions than the no cue conditions for both groups, with the AD group having longer "pause times" than the controls. Movement time was also longer for the no cue condition for both groups, with the AD subjects again having longer movement time than controls. AD subjects also made more submovements than controls in the no cue condition. This led the authors to suggest that AD subject's increased movement time reflected, at least in part, deficits in on-line reprogramming of the movement. Both groups had longer acceleration periods in the no cue condition and there was no difference in accuracy between the two groups. Overall, the authors concluded that AD subjects were able to use cues

to plan movements but that they were less efficient in their planning than were controls. They took the fact that the AD subjects had more procedural errors than controls (failing to complete the task in 20 seconds; moving to an incorrect target; lifting pen during movement; retracing or turning back on their own movements) in the no cue condition as evidence that cuing may be necessary to direct the behavior of AD subjects.

Although this study had the advantage of examining the kinematics of motor function in AD, the task used was quite complex. It involved a sequence of four movements and required the use of a pen, and the knowledge of its use (praxis). While the authors labelled the conditions "cued" and "uncued", this labelling is imprecise. There were, in fact, no "cues" provided about the probable location of the target. Rather, there was an "immediate target display condition" and a "delayed target display condition". Since there was no advance location cue, subjects would have had to attend to all possible locations prior to target display in both experimental conditions. In the immediate display condition ("cued"), after target onset, the subject could shift their attention to the displayed target and ignore other potential targets until this movement was completed. This process would be repeated until the end of the sequence. In the delayed target display condition ("uncued"), the subject had to initiate a movement in an unspecified direction while

continuing to attend to all potential target locations. Neither experimental condition provided advance information about the location or direction of the target for the movement. Thus, the paradigm of Bellgrove et al. (1997) cannot be interpreted within the framework of other cuing paradigms or within the conceptual framework on which most studies of cue-directed shifts of visual attention are based.

Visual Attention

Studies of shifting visual attention in AD have also been few. It has been suggested that damage to the basal forebrain cholinergic system (which innervates the prefrontal cortex, thalamus, and parietal lobes, and which are known to be involved in spatial attention) may be partially responsible for the attentional deficits noted in tests of sustained and spatial attention in AD (Lawrence & Sahakian, 1995). A study (using an adaptation of the Posner paradigm) in monkeys that were lesioned in the basal forebrain cholinergic system were found to have slowed RT for invalid trials (but not valid trials) in comparison to controls (Voytko, Olton, Richardson, Gorman, Tobin, & Price, 1994). Drugs that increase the level of acetylcholine (e.g., tacrine, nicotine) have been noted to improve performance on tests of attention in AD subjects. This has led to the hypothesis that damage to this system may serve to decrease the signal-to-noise ratio at the cortical level and decrease attentiveness to stimuli (Lawrence & Sahakian, 1995). Nicotine has been found to reduce the "costs" in

neurologically intact subjects (Murphy & Klein, 1998), suggesting that the cholinergic system may be associated with the disengagement of attention.

Stuss and colleagues have argued that there is an anterior attentional system, centred in the frontal lobe, which is responsible for the executive control of attention while a posterior attentional system, centred in the parietal lobe, is responsible for the spatial allocation of attention (Stuss, Shallice, Alexander, & Picton, 1995). Posner and colleagues have argued that the posterior parietal lobe is also associated with the disengagement of attention (see Posner & Petersen, 1990 for a review). The relationship between attentional deficits and AD which result in pathology to the frontal and parietal cortex has recently begun to be investigated, although the paradigms and dependent measures used have varied extensively.

Parasuraman, Greenwood, Haxby, and Grady (1992) combined the study of attentional shifts in AD with functional neuro-imaging. They examined both peripheral and central cue-directed shifts of visual attention in 15 subjects with mild to moderate AD and 15 matched controls, using a letter discrimination task. One of three types of cues (valid, invalid, or neutral) followed a 1 second warning signal (a plus sign) at various SOA's (200ms, 500ms, 2000ms). A target letter was then presented to either the right or left of a central fixation point. Subjects were to maintain fixation

until target presentation, and were to press a button, as quickly as possible, with either the left or the right index finger (depending on whether the letter was a vowel or a consonant). Eye movements were monitored for a subset of patients, using a mirror and video camera and all subjects were able to maintain fixation in the central cue condition. Regional resting cerebral metabolic rates for glucose were collected using positron emission tomography (PET) (and timed close to the time of the cuing study) for the superior parietal, inferior parietal, prefrontal, and occipital areas. The hand which was to respond to vowels and consonants, the three SOA's, and central or peripheral cues were randomized within each block. Results indicated that, although AD subjects were less accurate than controls, accuracy did not differ as a function of cue for either group. The AD and control subjects did not differ in RT benefits for either the peripheral or central cues at any SOA. The AD subjects had overall higher "costs plus benefits" for RT than controls but for central cues, this "costs plus benefits" difference was significant only at the 2000ms SOA, while for peripheral cues the AD subjects had greater "costs plus benefits" only at the 200ms SOA. The authors concluded that since both groups showed similar RT benefits, the initial shift of attention is not impaired in AD. Given the increased RT costs for AD subjects, however, they concluded that AD subjects are impaired in re-orienting or disengaging attention from an incorrect location.

The PET scan results revealed that the resting cerebral metabolic rates for glucose for the right superior parietal lobe were correlated moderately with RT "costs plus benefits" for both peripheral cues (-0.54) and central cues (-0.42) and reflected the fact that the AD subjects with greater hypometabolism to the right superior parietal lobe (compared with the left superior parietal lobe) had higher "costs plus benefits" than those AD subjects with greater left than right hypometabolism. Correlations were nonsignificant for the other areas measured. The authors took this as evidence supporting the hypothesis that right superior parietal lobe pathology is related to deficits in the disengagement of spatially directed attention in AD, although they acknowledged that causality cannot be inferred from their study.

Oken, Kishiyama, Kaye, and Howieson (1994) examined covert orienting of spatial attention in 10 patients with probable AD (mean MMSE = 16.4; range = 11 to 25), 10 healthy elderly controls, and 15 young adult controls. The young controls were tested twice, once in a regular manner and once after being given the drug Diphenhydramine (which has both antihistaminergic and anti-cholinergic effects) to reduce alertness. The paradigm consisted of a circle or a square (square was the catch trial) being presented to the left or the right of fixation following a 100ms central cue (valid arrow, invalid arrow, neutral plus sign). The SOA was 800ms. If the stimulus was a circle, subjects were instructed to push

a hand-held button with the thumb of the right hand as quickly as possible. If the stimulus was a square, they were instructed to do nothing. As is usually the case, the subjects were told that most of the time the cue would be valid but sometimes it would not. The authors noted the AD subjects seemed to have difficulty understanding this concept (although no examples of this were provided). Eye movements were monitored via an infrared-corneal reflection system. Only correct responses with reaction times between 100ms and 1500ms were analyzed. Analyses used the means of the median RT's in each condition to compare group performance. Results indicated that, after ingesting the drug, the young subjects showed a slowing of RT of about 10% but showed no difference in their "costs plus benefits" (i.e., no disproportionate slowing of RT across conditions). The AD subjects had greater "costs plus benefits" than the elderly control subjects due to a higher RT for invalid cues. The authors concluded that the AD subjects had deficits in shifting attention compared to the elderly controls, but that this deficit was not likely due to deficits in alertness produced by alterations in acetylcholine levels. The authors did acknowledge, however, that the effects of an acute blocking of a neurotransmitter are different than the effects of neurotransmitter decrease associated with neural degeneration. Of note, these authors interpreted the increased RT in the invalid condition as an indication of a deficiency in the shift of attention in AD. While their result is

consistent with the findings of previous research (e.g., Parasuraman et al., 1992), others have interpreted this finding as a deficit in the disengagement of attention (e.g., Parasuraman et al., 1992) and have associated a shift of attention with the measure of benefits. Thus, most studies support the argument that the process of shifting attention is intact in AD while the disengagement of attention process may be defective.

A study by Wright, Cremona-Meteyard, Geffen, and Geffen (1994) also provides support for intact shifting of attention in AD but did not specifically investigate the disengagement of attention. They investigated the covert orienting of visual attention in 11 AD subjects and 11 matched controls using a task in which a horizontal bar served as a target while central cues (valid, invalid - left or right arrows; neutral - plus sign) were presented with a SOA of 1100ms. The task was to press a button with the dominant index finger when the target was illuminated. The cues remained on for the duration of the trial to remove any need for the subjects to use memory to locate the target. Subjects were provided feedback on their performance on the computer screen in which either their exact RT was shown or an error message indicated that the RT was too fast (< 100ms) or too slow (> 900ms). There was also a no-go trial where a vertical line was presented and the task was to inhibit a response. Results revealed that AD subjects had more errors than controls including missing targets (which happened

more for neutral central cues) and anticipatory responses, and responding more often to the no-go trials compared with controls (23% vs 3%, respectively). However, AD subjects showed an RT advantage to valid cues compared with neutral cues (i.e., benefit) which suggested that they had no deficit in the shifting of attention. They had greater "benefits" than the elderly control group which was likely due to the long RT's in the neutral conditions. There were no "costs". AD subjects had the longest RT to neutral cues (compared with the valid and invalid cues) which the authors suggested was indicative of a deficit in dividing attention across the complete visual space. Unfortunately, this study did not calculate and present "costs plus benefits", which may have been a better measure for the AD group given that the results of the neutral cue condition made the costs and benefits difficult to interpret independently (eg., Jonides & Mack, 1984). While not explicitly stated, it appears that RT responses greater that 900ms were not used, suggesting that valuable data may have been lost.

Although the studies of Parasuraman et al. (1992) and Oken et al. (1994) can be taken as support for an impairment of the disengagement process in AD, a study by Caffara, Riggio, Malvezzi, Scaglioni, and Freedman (1997) did not support this. Of note, they used only valid and invalid cues (80%, 20% probability, respectively) in a study of the orienting of attention in 7 subjects with probable AD and 10

control subjects. Following the presentation of a central fixation point that remained on for 1000ms, an informational cue (a left or right pointing arrow) was presented and subjects were instructed to attend to the cued side. After an SOA of either 100ms or 800ms, a target (square) was presented to the left or right of fixation. Subjects were instructed to press a key with the right index finger when the target was perceived. Results indicated that RT was faster for controls than for AD subjects but that both groups had faster RT to valid than invalid cues at both SOA's. There was no group by cue type interaction, leading the authors to suggest that there are no differences in the disengagement of attention in AD and controls. Unfortunately, these authors did not use or report on any responses that were greater than 1500ms. Although they considered these to be errors, they may, in fact, have discarded meaningful data.

As is evident from the few studies that have examined motor behavior and cue directed shifts of attention in AD, there appears to be reason to suggest that persons with AD have some deficits in complex motor functioning and with the disengagement of attention. Most studies suggest, however, that AD subjects are not impaired on the initial shifts of attention.

CURRENT STUDY

The only kinematic study of arm movements by AD subjects that claimed to investigate "cue directed" shifts of attention

on motor programming did not use cues in a manner similar to that of most research on the cognitive process of attentional shifts (Bellgrove et al., 1997). However, the current study paired the traditional cuing paradigm adapted from Posner (1980) with kinematic analysis of visually directed limb movement. By comparing the performance of AD patients and healthy elderly control subjects we hoped to determine whether AD patients have distinct deficits in shifting of attention and/or motor response planning. By examining detailed aspects of the movement trajectory in different cuing conditions we also hoped to contribute to the understanding of the manner in which visual attention and motor function are related and/or impaired in AD.

HYPOTHESES

Cue Effects:

The AD group was expected to show an advantage of informational cues that were determined to be valid in comparison to those that were determined to be invalid for both paradigms. The AD group was expected to show an advantage of exogenous cues over endogenous cues (as have both the young and elderly subject groups).

The AD group was expected to have larger "costs plus benefits" for the Exogenous than the Endogenous Paradigm (due to the reported deficits in the disengagement of attention process).

AD vs Elderly Controls:

The AD subject group was expected to have slower RT, longer MT, lower PV, and equivalent accuracy compared with the elderly control group. Given that the literature has suggested that AD subjects have greater difficulty with on-line reprogramming of movements than elderly controls, it was expected that they would have a longer percentage of the movement take place in the deceleration phase compared with elderly controls. This would allow them to use feedback to make terminal corrections in order to intercept the target accurately.

The "costs plus benefits" was expected be greater for the AD group than for the elderly control group for both paradigms. This was expected due to their greater deficits in the disengagement of attention process and to possible greater deficits in response programming than health elderly individuals.

METHOD

Subjects

Subjects were 14 patients (7 females; 7 males) who had been assessed by a neurologist or a geriatrician in the Memory Disability Clinic of the Queen Elizabeth II Health Sciences Centre and who met the criteria for probable Alzheimer's Disease as established by the DSM-IV and the NINCDS-ADRDA Working Group criteria (McKhann et al., 1984). The breakdown of gender between the AD and the elderly control group

differed significantly (chi-square = 4.54, $p < .05$) with the elderly control group having a greater number of females and a fewer number of males compared with the AD group. All AD subjects were right handed as determined by same criteria as healthy young and elderly subjects. None had physical impairments that would interfere with the movement of the right arm and hand (as judged by the clinical staff of the Memory Disorders Clinic and the experimenter). All were judged (by knowledgeable clinical staff) to have sufficient cognitive abilities to allow them to perform the task. All had vision that was either normal or corrected to allow them to be able to see the computer screen with no difficulty (as indicated by a brief screening of vision that involved the subject reading the numbers on the computer screen that was used for the experiments and pointing to each of the numbers upon command). Informed consent was obtained from all subjects (and in 1 case the guardian) in accordance with procedures approved by the Queen Elizabeth II Health Sciences Centre Research Ethics Committee. Eight of the subjects were taking part in double blind drug trials for potential medications in the treatment of AD. Thus, some subjects may have been receiving medication while others may have been on the placebo arm of the trial.

Cognitive status was evaluated using the Mini Mental State Exam (MMSE) (Folstein, Folstein, & McHugh, 1975) on the day of the experiment for all subjects. The MMSE is a standardized 11 item cognitive screening examination which

assesses orientation, immediate and delayed recall of three words, attention (spell WORLD backwards), calculation, language (comprehension, repetition, writing) and visual construction (Folstein et al., 1975). Scores can range from 0 to 30 with higher scores indicating a higher level of cognitive functioning. Scores from 18 to 23 are considered to indicate the presence of at least mild dementia, while those under 18 are considered to indicate the presence of moderate dementia. In the current study, the MMSE scores were used as an estimate of the stage of AD.

Despite the fact that only two subjects had MMSE scores below 18 (i.e., 17/30), only 10 of the 14 AD subjects were able to complete both target paradigms. Specifically, four subjects were unable to respond in accordance with the task instructions during the endogenous target paradigm when provided with the standard instruction procedure and ample opportunities for practice. This was not a function of the order of task presentation as this order varied randomly and was either the first or the second task attempted for two of the four subjects. As a result, the AD group was divided into two subgroups for the purpose of some analyses. One group included those who could complete both experiments [AD(A), n = 10] while the other included those who completed only the exogenous paradigm [AD(B), n = 4]. Only one subject in the AD(A) group had an MMSE score under 20 while all others with an MMSE score under 20 were in the AD(B) group.

The mean MMSE score for the AD(A) group was 23.8 (SD = 3.4) with a range of 17 to 29. The mean MMSE for the AD(B) group was 18.3 (SD = 0.96) with a range of 17 to 19. Mean age for the AD(A) group was 69.2 (SD = 9.4) with a range of 47 to 80 years. Mean age for the AD(B) group was 71.8 (SD = 8.2) with a range of 67 to 81 years. Mean level of education for the AD(A) group was 12.5 (SD = 2.1) with a range of 10 to 16 years. Mean level of education for the AD(B) group was 12.5 (SD = 5.7) with a range of 4 to 16 years. Thus, mean age and level of education did not differ between the two AD subject groups nor did they differ between the total AD group and the elderly control group (all p values > .05). The demographic data for these groups are illustrated in Table 6.1. The specific details for the AD subjects are presented in Table 6.2.

Table 6.1

Young, Elderly Control, and AD Subject Demographics Table

	Young	Elderly Controls	AD(A)	AD(B)
N	15	15	10	4
M/F	7/8	2/13	6/4	2/2
Mean Age	28.6	69.5	69.2	72.8
Range	24-39	58-77	47-80	67-81
Mean Educ	17.7	13.4	12.1	12.5
Range	13-20	6-18	11-16	4-16
Mean MMSE	____	29.9	23.8	18.2
Range	____	29-30	17-29	17-19

Table 6.2

Alzheimer Subject Demographics

Subject	Age	Sex	Educ	MMSE	Drug Trial
Group A					
1	47	M	11	26	Y
2	64	M	11	20	Y
3	65	M	12	24	Y
4	67	F	14	23	N
5	71	M	15	24	Y
6	71	F	14	23	Y
7	75	F	15	27	N
8	76	M	11	29	N
9	76	M	10	25	Y
10	80	F	14	17	Y
Group B					
11	67	F	14	18	N
12	67	M	16	17	Y
13	76	F	16	19	N
14	81	M	04	19	N

Procedure

The procedure was identical to that described previously although a greater number of practice trials (usually 20 or 30 trials) were typically required for the AD subjects to learn the task. The two blocks of 60 experimental trials were presented for most subjects. Three of the 4 subjects in the AD(B) group and 1 subject in the AD(A) group were only able to complete 1 trial block of each experimental condition due to fatigue.

Analyses

The same procedure for statistical analyses were followed as were described in chapter 3. Since the AD group was divided into two subgroups, two separate analyses were conducted on the AD data. The first analysis was conducted on the AD(A) group (i.e., using only the 10 AD subjects who were able to complete both experimental paradigms). The second analysis compared the elderly control group and the AD(A) group. The third analysis compared the two subgroups of AD subjects [i.e., AD(A) vs AD(B)].

ANALYSIS 1

Analysis 1 was a 3 (Cue Condition - Valid, Invalid, Neutral) X 2 (Target Type - Exogenous, Endogenous) within subjects (cue condition and target type) repeated measures MANOVA using SAS General Linear Models procedures. The dependent measures were reaction time (RT), time from movement initiation to completion (MT), peak velocity (PV), percent

deceleration and resultant error.

A series of planned paired comparisons using the least squares adjusted means was conducted to examine the differences in "costs plus benefits" (i.e., invalid minus valid) for each of the dependent variables.

RESULTS

AD(A) Group

The MANOVA revealed multivariate main effects of cue condition, $F(8, 32) = 4.34$, $p < .002$. There was a marginal multivariate effect of target type, $F(4, 6) = 3.81$, $p = .07$ and no multivariate cue condition by target type interaction, $F(8, 32) = 1.36$, $p = .25$. The MANOVA was followed up by a univariate mixed model Analysis of Variance (ANOVA) for each of the five dependent variables.

Reaction Time (See Figure 6.1)

Results of the univariate mixed model ANOVA indicated that there was a univariate main effect of cue condition for RT, $F(2, 18) = 20.91$, $p < .0001$. Comparison of least squared adjusted means (collapsing across target type) indicated that the mean for the valid condition ($M = 813.58$ms, $SE = 25.7$ms) was significantly faster than that of the invalid ($M = 1039.3$, $SE = 42.9$; $t = 4.51$, $p < .0005$) and the neutral ($M = 1092.4$ms, $SE = 43.3$ms; $t = 5.53$, $p < .0001$) conditions. The neutral and invalid conditions did not differ from one another ($t = 0.87$, $p = .40$). There was a univariate main effect of target type, $F(1,9) = 12.38$, $p < .01$. A comparison of the least squares

adjusted means revealed that the RT for the Exogenous Target Paradigm (*M* = 814.6ms, *SE* = 68.3) was significantly faster than that of the Endogenous Paradigm (*M* = 1148.9, *SE* = 67.9, *t* = 3.47, *p* < .01). There was no significant cue condition by target type interaction, *F* (2, 18) = 0.34, *p* = .71. The "costs plus benefits" for the Exogenous Paradigm was 226.1ms (*t* = 4.75, *p* < .0002) and was 225.4ms for the Endogenous Paradigm (*t* = 4.77, *p* < .0002).

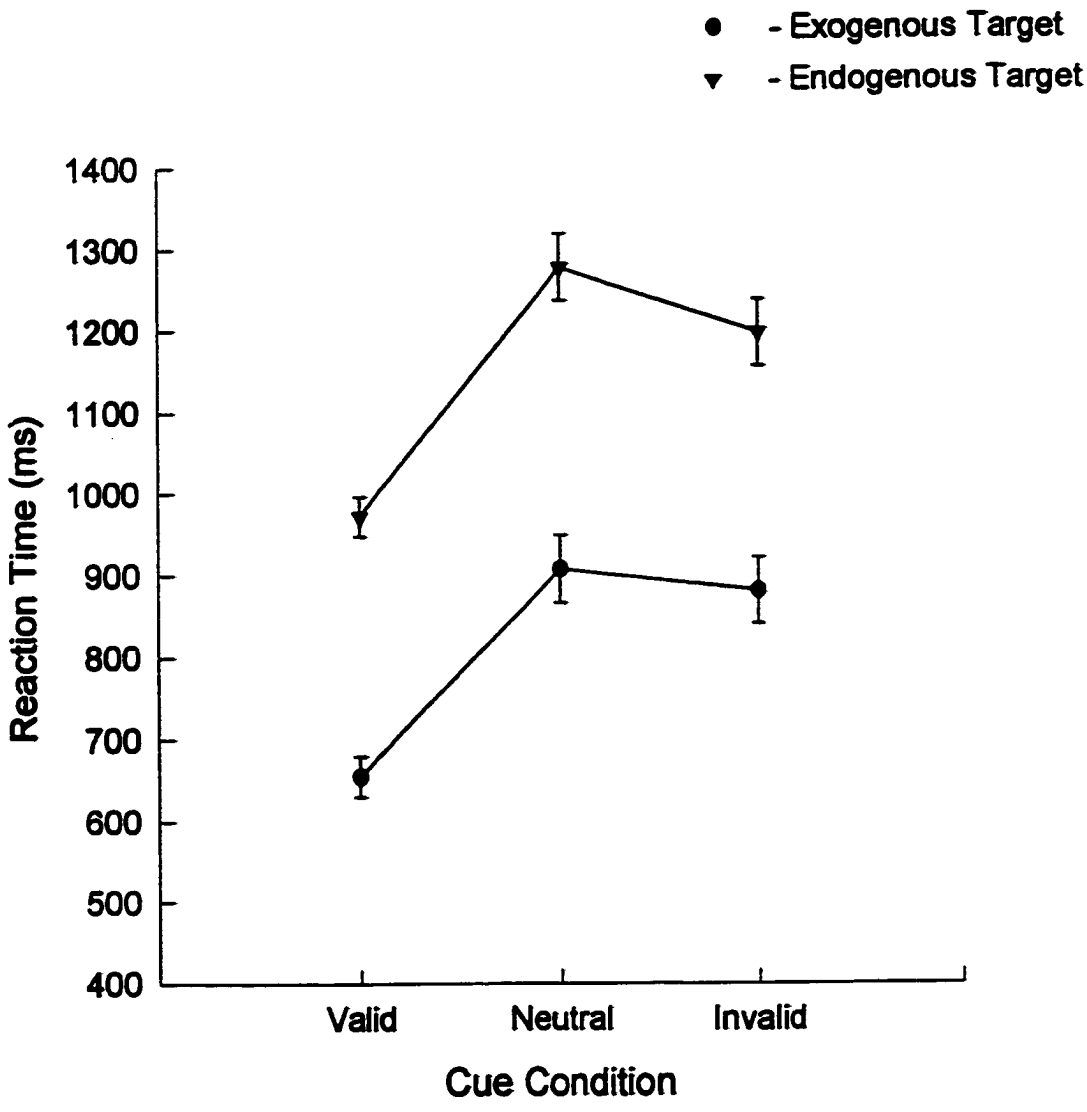


Figure 6.1 - Reaction time as a function of cue condition and target paradigm. The error bars represent the standard error of the least squares means.

Movement Duration (See Figure 6.2)

Results of the univariate mixed model ANOVA indicated that there was a main effect of cue condition for MT, $F(2, 18) = 5.97$, $p < .01$. Comparison of least squared adjusted means (collapsing across target type) indicated that the mean for the valid condition (M = 776.3ms, SE = 10.7ms) was significantly faster than that of the invalid condition (M = 846.9, SE = 17.8; $t = 3.41$, $p < .005$), and did not differ from the neutral (M = 795.5ms, SE = 17.9ms; $t = 0.92$, $p = .37$) condition. The invalid and the neutral conditions differed only marginally ($t = 2.03$, $p = .06$). There was a marginally significant univariate main effect of target type, F (1,9) = 3.50, $p = .09$. A comparison of the least squares adjusted means revealed that the MT for the Exogenous Paradigm (M = 773.4ms, SE = 25.3ms) was marginally faster than that of the Endogenous Paradigm (M = 839.1ms, SE = 25.1ms, $t = 1.84$, $p = .09$). There was also a marginally significant cue condition by target type interaction, F (2, 18) = 2.87, $p = .08$. This interaction was due to the difference in patterns between the two target types. There was no difference among cue conditions for the Exogenous Paradigm (all p values > .05). However, for the Endogenous Paradigm, the valid cue condition (M = 789.2, SE = 15.5ms) was significantly shorter than the invalid condition (M = 910.6ms, SE = 26.2ms; $t = 3.99$, $p < .001$) and the invalid condition was significantly shorter than the neutral condition (M = 817.5ms, SE = 26.4ms; $t = 2.54$, $p <$

.05). The "costs plus benefits" for the Endogenous Paradigm was significant (121.4ms, $p < .001$) while those for the Exogenous Paradigm were not significant (19.8ms, $p = .52$).

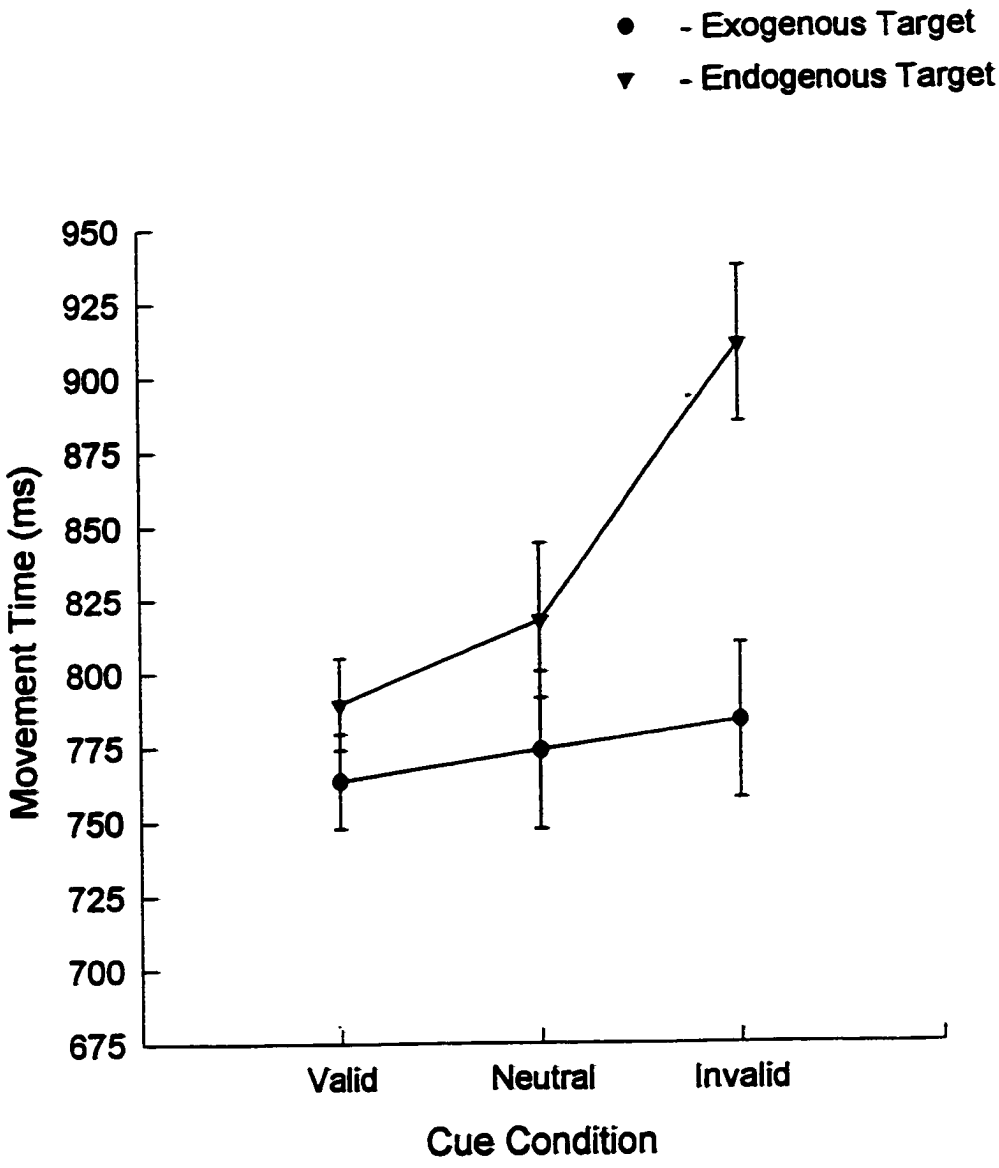


Figure 6.2 - Movement time as a function of cue condition and target paradigm. The error bars represent the standard error of the least squares means.

Peak Velocity (See Figure 6.3)

Results of the univariate mixed model ANOVA indicated that there was no main effect of cue condition for PV, $F(2, 18) = 2.20$, $p = .14$, or target type, $F(1,9) = 1.98$, $p = 0.19$. There was a marginally significant cue condition by target type interaction, $F(2, 18) = 3.12$, $p = .07$. This interaction was due to the difference in patterns between the two target types. There was no difference among cue conditions for the Exogenous Target Paradigm (all p values > .05). However, for the Endogenous Target Paradigm, the valid cue condition (M = 66.2cm/sec, SE = 0.37cm/sec) was significantly higher than the invalid condition (M = 64.2cm/sec, SE = 0.63cm/sec; $t = 2.77$, $p < .05$) and the neutral condition (M = 63.9cm/sec, SE = 0.64cm/sec, $t = 3.00$, $p < .01$). The "costs plus benefits" for the Endogenous Target Paradigm was 2.0cm/sec ($p < .05$), while there were no "costs plus benefits" for the Exogenous Target Paradigm ($p = .93$).

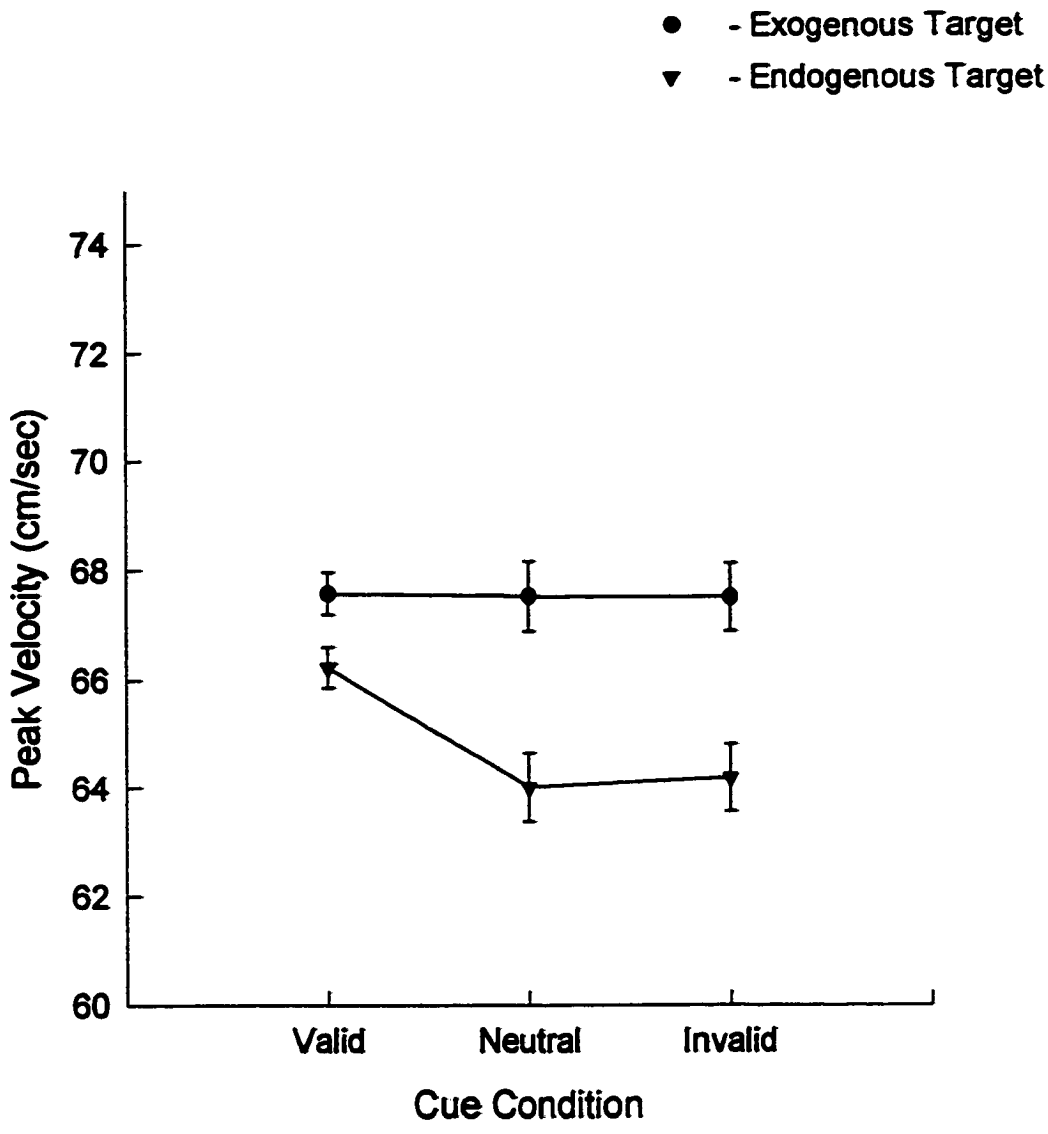


Figure 6.3 - Peak velocity as a function of cue condition and target paradigm. The error bars represent the standard error of the least squares means.

Percent Deceleration (See Figure 6.4)

Results of the univariate mixed model ANOVA indicated that there was no main effect of cue condition for percent deceleration, $F(2, 18) = 0.64$, $p = .54$, or target type, $F(1,9) = 0.09$, $p = .77$. There was no cue condition by target type interaction, $F(2, 18) = 0.29$, $p = .75$. There were no significant "costs plus benefits" for either target paradigm.

Resultant Error (See Figure 6.5)

Results of the univariate mixed model ANOVA indicated that there was no main effect of cue condition for resultant error, $F(2, 19) = 0.18$, $p = .84$, or target type, $F(1,9) = 0.23$, $p = .64$. There was no cue condition by target type interaction, $F(2, 19) = 0.16$, $p = .85$. There were no significant "costs plus benefits" for either target paradigm.

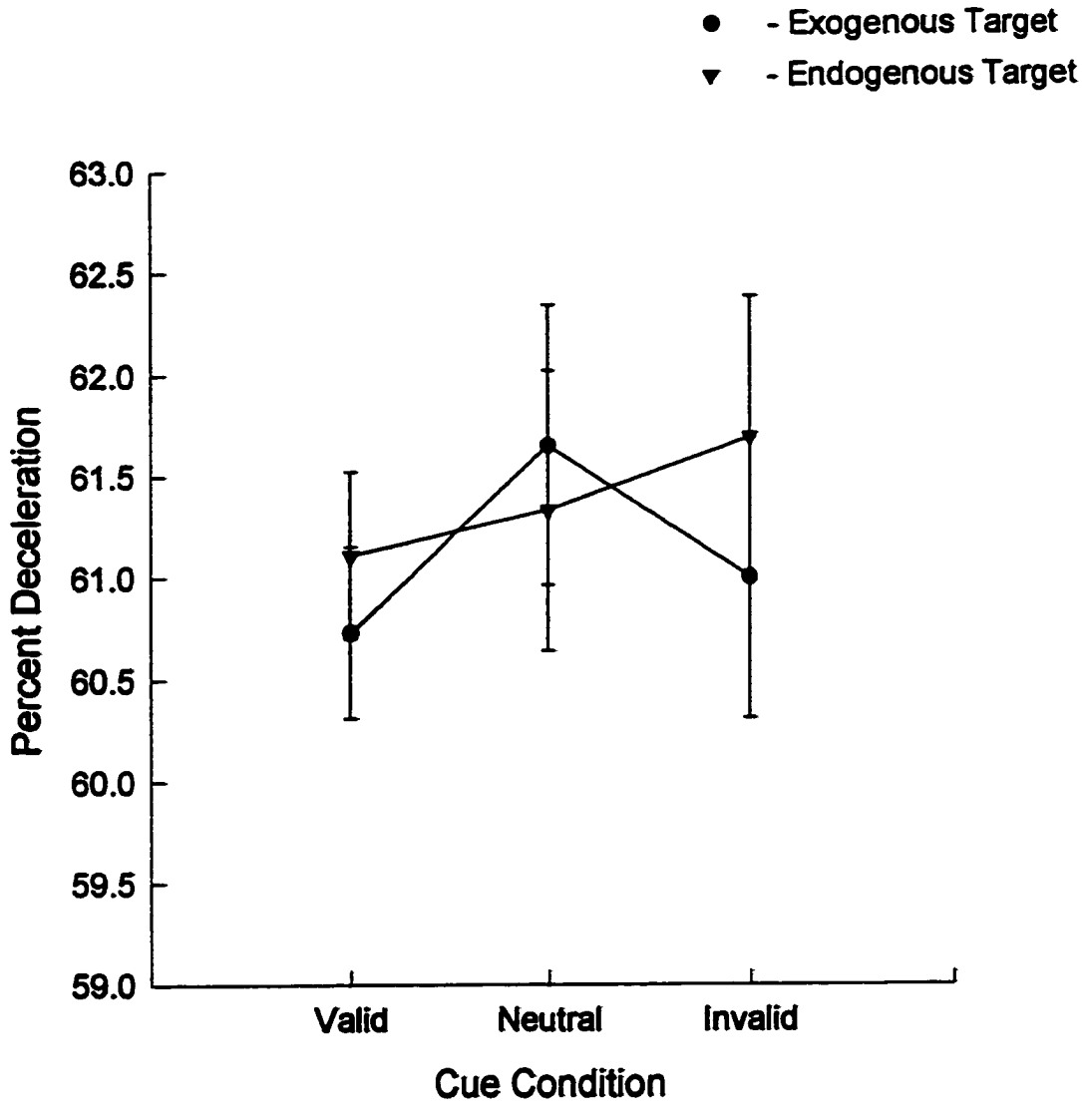


Figure 6.4 - Percent deceleration as a function of cue condition and target paradigm. The error bars represent the standard error of the least squares means.

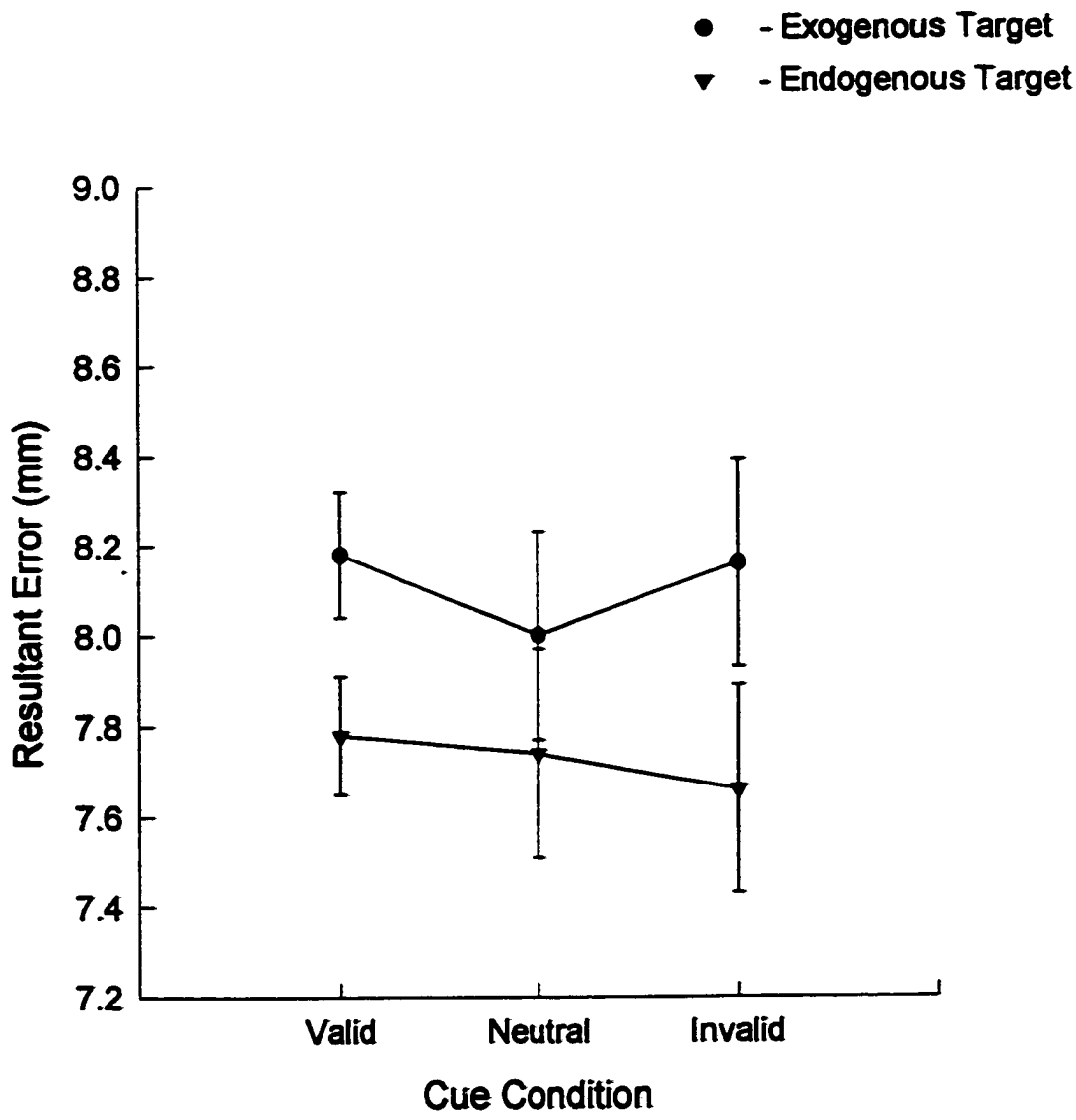


Figure 6.5 - Resultant error as a function of cue condition and target paradigm. The error bars represent the standard error of the least squares means.

ANALYSIS 2

Analysis 2 was a 3 (Cue Condition - Valid, Invalid, Neutral) X 2 (Target Type - Exogenous, Endogenous) X 2 (Group - Elderly Control, AD) repeated measures MANOVA using SAS General Linear Models procedures. Group was a between subjects factor while cue condition and target type were within subject factors. The dependent measures were reaction time (RT), time from movement initiation to completion (MT), peak velocity (PV), percent deceleration and resultant error.

A series of planned paired comparisons using the least squares adjusted means was conducted to examine the differences in "costs plus benefits" (i.e. Invalid minus Valid) for each of the dependent variables.

RESULTS

Elderly Controls Vs. AD(A)

The MANOVA revealed multivariate main effects of group, $F(4, 20) = 4.07$, $p < .02$, cue condition $F(8, 88) = 8.83$, $p < .0001$, and target type $F(4, 20) = 8.75$, $p < .0005$. In addition, there was a multivariate group by condition interaction, $F(8, 88) = 3.78$, $p < .0005$, and a multivariate group by target type interaction, $F(4, 20) = 3.19$, $p < .05$. The condition by target type interaction did not reach statistical significance, $F(8, 88) = 1.72$, $p = .10$, and only a marginal multivariate group by condition by target type interaction, $F(8, 88) = 2.03$, $p = .05$. The MANOVA was followed up by a univariate mixed model Analysis of Variance (ANOVA)

for each of the five dependent variables.

Reaction Time (See Figure 6.6)

Results of the univariate mixed model ANOVA indicated that there was a main effect of group for RT, $F(1, 23) = 10.19$, $p < .005$. Comparison of least squared adjusted means (collapsing across cue condition and target type) indicated that the AD(A) subjects had an average RT that was 357ms slower than that of the elderly controls (elderly controls = 624.8ms, SE = 75.8ms; AD = 981.8ms; SE = 95.9ms, $t = 3.15$, $p < .005$) (collapsed across cue condition and target type). There was a main effect of cue condition, $F(2,46) = 45.49$, $p < .0001$. Comparisons of the least squares adjusted means (collapsing across target type and group) indicated that the neutral cue condition had the slowest RT (M = 879.9ms; SE = 20.1ms), followed by the invalid condition (M = 842.0ms; SE = 19.9ms) and the valid condition (M = 687.9ms; SE = 11.8ms). The valid condition was significantly faster than the invalid condition ($t = 6.63$, $p < .0001$) and the neutral condition ($t = 8.23$, $p < .0001$). The invalid and neutral conditions were equivalent ($t = 1,33$, $p = .19$).

There was a group by cue condition interaction, $F(2, 46) = 9.58$, $p < .0003$. While both groups had the same pattern of results (i.e., valid faster than invalid; valid faster than neutral; invalid = neutral), the magnitude of differences among the cue conditions was greater for the AD than the elderly control group (collapsing across target type). There

was also a group by target type interaction $F(1, 23) = 10.44$, $p < .005$. This was due to there being a greater difference between the two target paradigms (collapsed across cue condition) for the AD(A) group (RT difference = 334.3ms, $t = 5.28$, $p < .0001$) than for the elderly subject group (RT difference = 77.1ms, $t = 1.54$, $p = 0.13$). There was no group by condition by target type interaction, $F(2, 47) = 1.07$, $p = .35$.

"Costs plus benefits" for the AD group for the Exogenous Paradigm was 224.2ms compared with 101.5ms for the elderly controls and this difference was statistically significant ($F = 5.84$, $p < .02$). While "costs plus benefits" was significantly greater for the AD group than for the elderly control group in the Endogenous Paradigm (225.3ms compared with 63.4ms), ($F = 8.09$, $p < .005$), the "costs plus benefits" did not differ between the two target paradigms for the AD subjects ($F = 0.14$, $p = .71$).

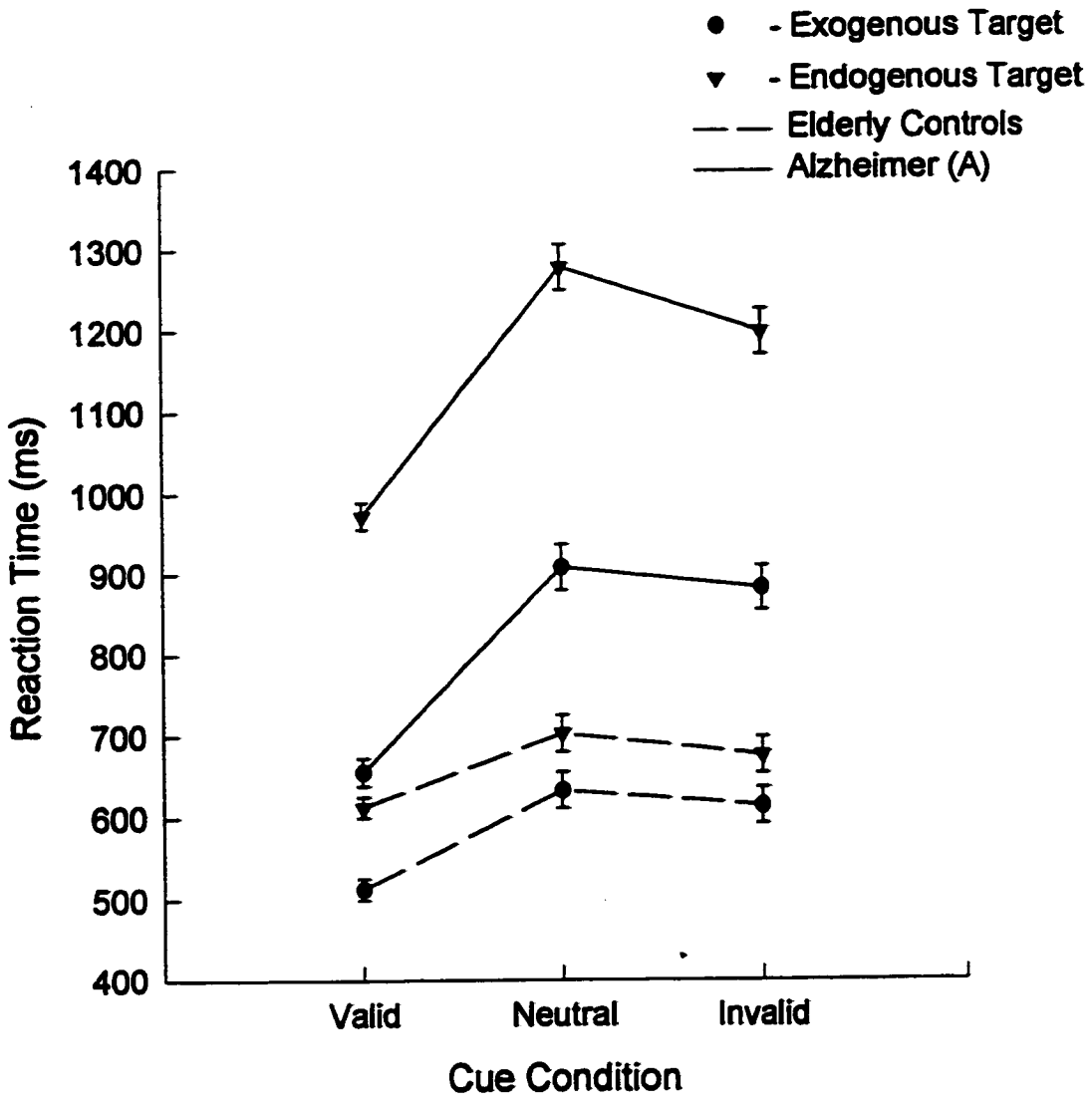


Figure 6.6 - Reaction time as a function of cue condition, target paradigm and group. The error bars represent the standard error of the least squares means.

Movement Duration (See Figure 6.7)

Results of the univariate mixed model ANOVA indicated that there was a main effect of group for MT, $F(1, 23) = 4.80$, $p < .05$. Comparison of the least squares adjusted means (collapsing across target type and cue condition) indicated that the AD subjects had an average overall MT that was 72ms slower than that of the elderly controls (elderly controls = 734.2ms, SE = 22.3ms; AD = 806.3ms, SE = 28.3ms; $t = 2.16$, $p < .05$).

There was also a group by cue condition interaction, $F(2, 46) = 6.99$, $p < .002$. There were no significant differences among the cue conditions for the elderly controls. However, for the AD group, MT was 70.6ms faster for the valid than the invalid conditions ($t = 5.15$, $p < .0001$), and was 51.4ms faster for the neutral than the invalid condition ($t = 3.08$, $p < .005$). The neutral and valid conditions did not differ ($t = 1.39$, $p = .17$) (collapsing across target type).

There was no group by target type interaction, $F(1, 23) = 1.63$, $p = .21$ but there was a group by cue condition by target type interaction, $F(2, 46) = 3.78$, $p < .05$. This resulted from different patterns between the two groups in the two target types. There were no significant differences in MT as a function of cue validity for the elderly controls for either the Exogenous or Endogenous Target Paradigms. There were, however, significant differences in MT as a function of cue condition for the AD subjects in the Endogenous Target

Paradigm (valid shorter than invalid, $t = 6.17$, $p < .0005$; neutral shorter than invalid, $t = 3.87$, $p < 0.005$; valid shorter than neutral, $t = 1.43$, $p = .16$). This, in turn, reflected the fact that the AD subjects demonstrated a particular elevation of MT (i.e., "costs") for the invalid cue condition of the Endogenous Target Paradigm.

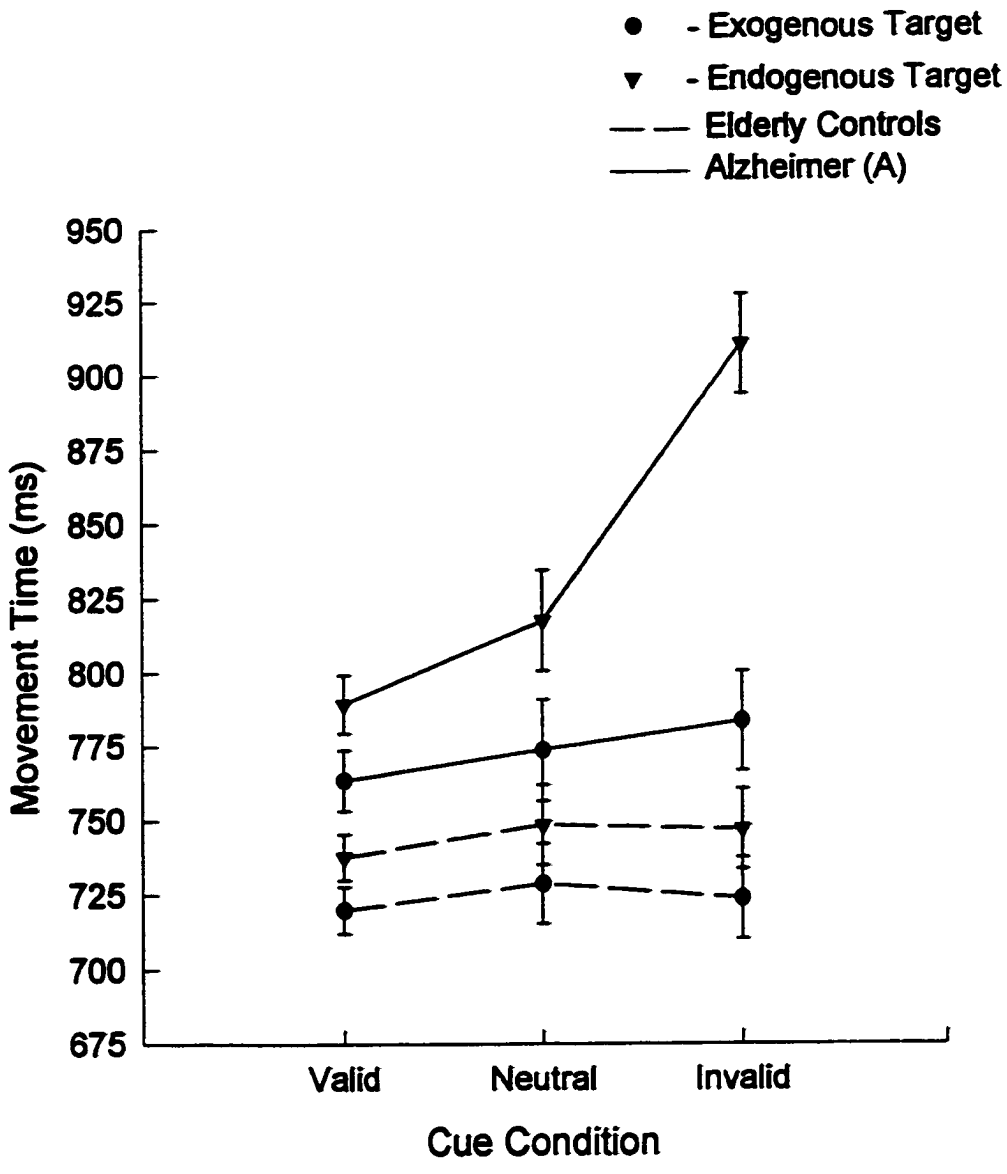


Figure 6.7 - Movement time as a function of cue condition, target paradigm and group. The error bars represent the standard error of the least squares means.

Peak Velocity (See Figure 6.8)

Results of the univariate mixed model ANOVA revealed a marginal effect of group for PV, $F(1, 23) = 4.11$, $p = .05$. Comparison of the least squared adjusted means (collapsing across target paradigm) indicated that the AD subjects had an average PV that was 7.1cm/sec lower than that of the elderly control subjects, and this difference was marginally significant (elderly controls = 73.3cm/sec, SE = 2.7cm/sec; AD = 66.2cm/sec, SE = 3.4cm/sec, $t = 2.00$, $p = 0.05$).

There was no univariate group by condition interaction, $F(2, 47) = 1.04$, $p = .37$, no group by target type interaction, $F(1, 23) = 0.01$, $p = .93$, and no group by condition by target type interaction, $F(2, 49) = 1.71$, $p = .19$. A priori planned comparisons indicated there was significant "costs plus benefits" for the AD subjects (2.0cm/sec; $t = 3.37$, $p < .005$) for the Endogenous Target Paradigm (with movements to validly cued targets having higher PV than those to invalidly cued targets) but there were no other significant "costs plus benefits".

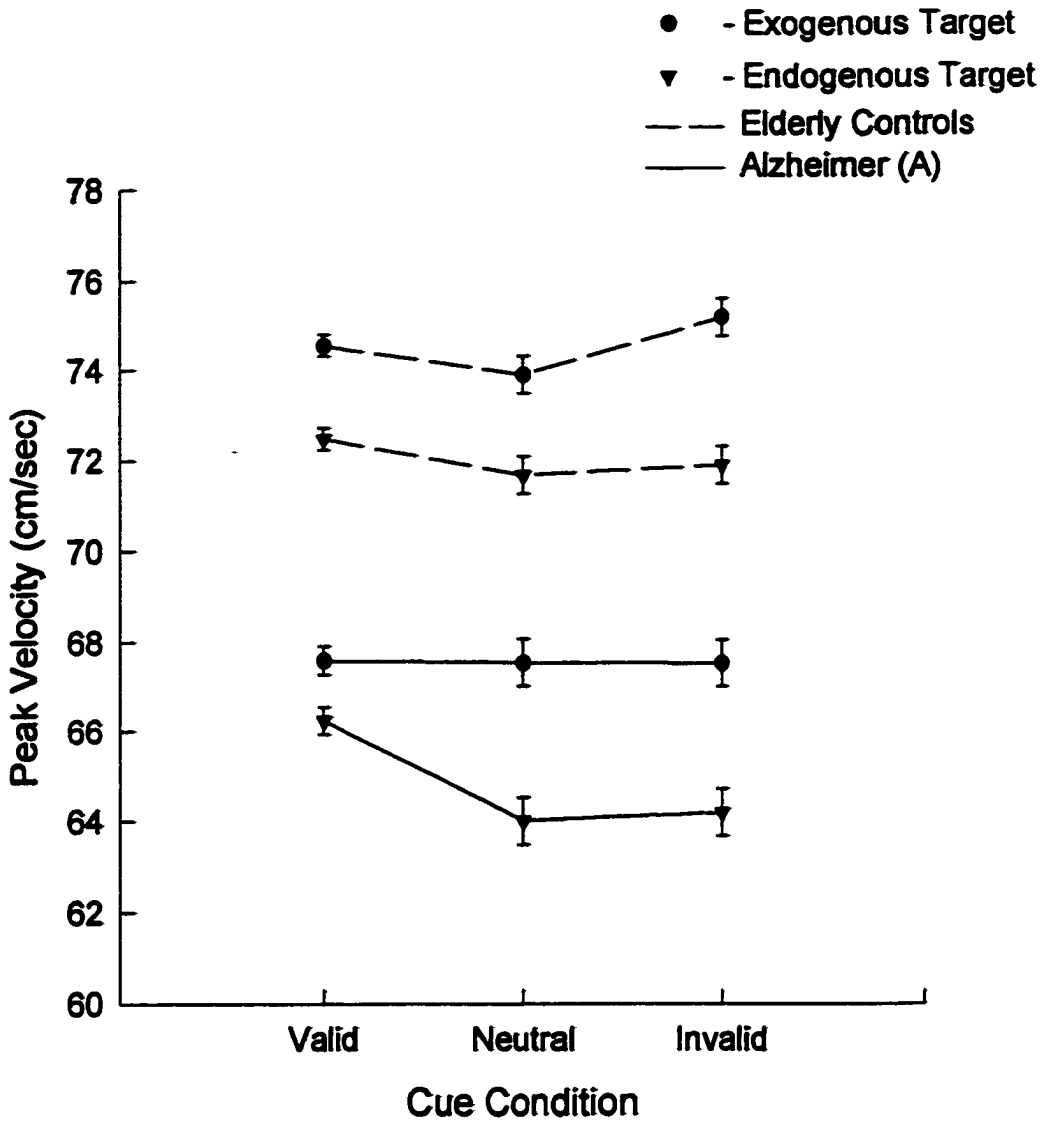


Figure 6.8 - Peak velocity as a function of cue condition, target paradigm and group. The error bars represent the standard error of the least squares means.

Percent Deceleration (See Figure 6.9)

Results of the univariate mixed model ANOVA revealed no univariate main effect of group for percent deceleration, $F(1, 23) = 1.84$, $p = .19$. There was no univariate group by condition interaction, $F(2, 47) = .23$, $p = .79$, no univariate group by target type interaction, $F(1, 23) = 0.70$, $p = .41$ and no univariate group by condition by target type interaction, $F(2, 47) = 0.49$, $p = .61$. There were no significant "costs plus benefits" for either group for either target paradigm.

Resultant Error (See Figure 6.10)

The results of the univariate mixed model ANOVA revealed no main effect of group for resultant error, $F(1, 23, = 0.26)$, $p = .61$. There was no group by condition interaction, $F(2, 48) = .16$, $p = .85$, no group by target type interaction, $F(1, 23) = 0.37$, $p = .55$, and no group by condition by target type interaction, $F(2, 48) = 0.28$, $p = .77$. Mean errors among the three cue conditions and two target paradigms ranged from 8.1mm to 8.5mm for the elderly controls and ranged from 7.7mm to 8.2mm for the AD(A) group.

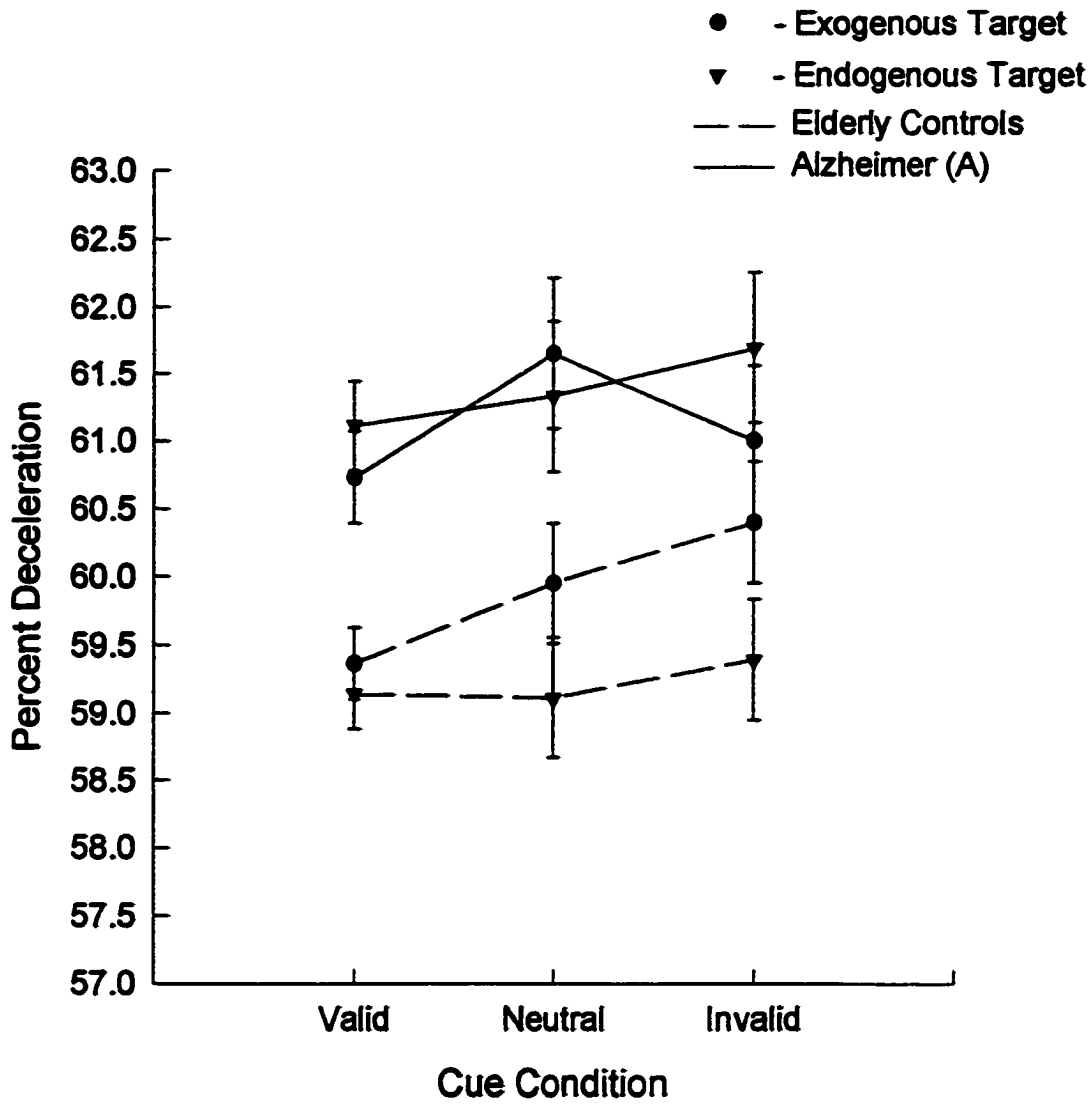


Figure 6.9 - Percent deceleration as a function of cue condition, target paradigm and group. The error bars represent the standard error of the least squares means.

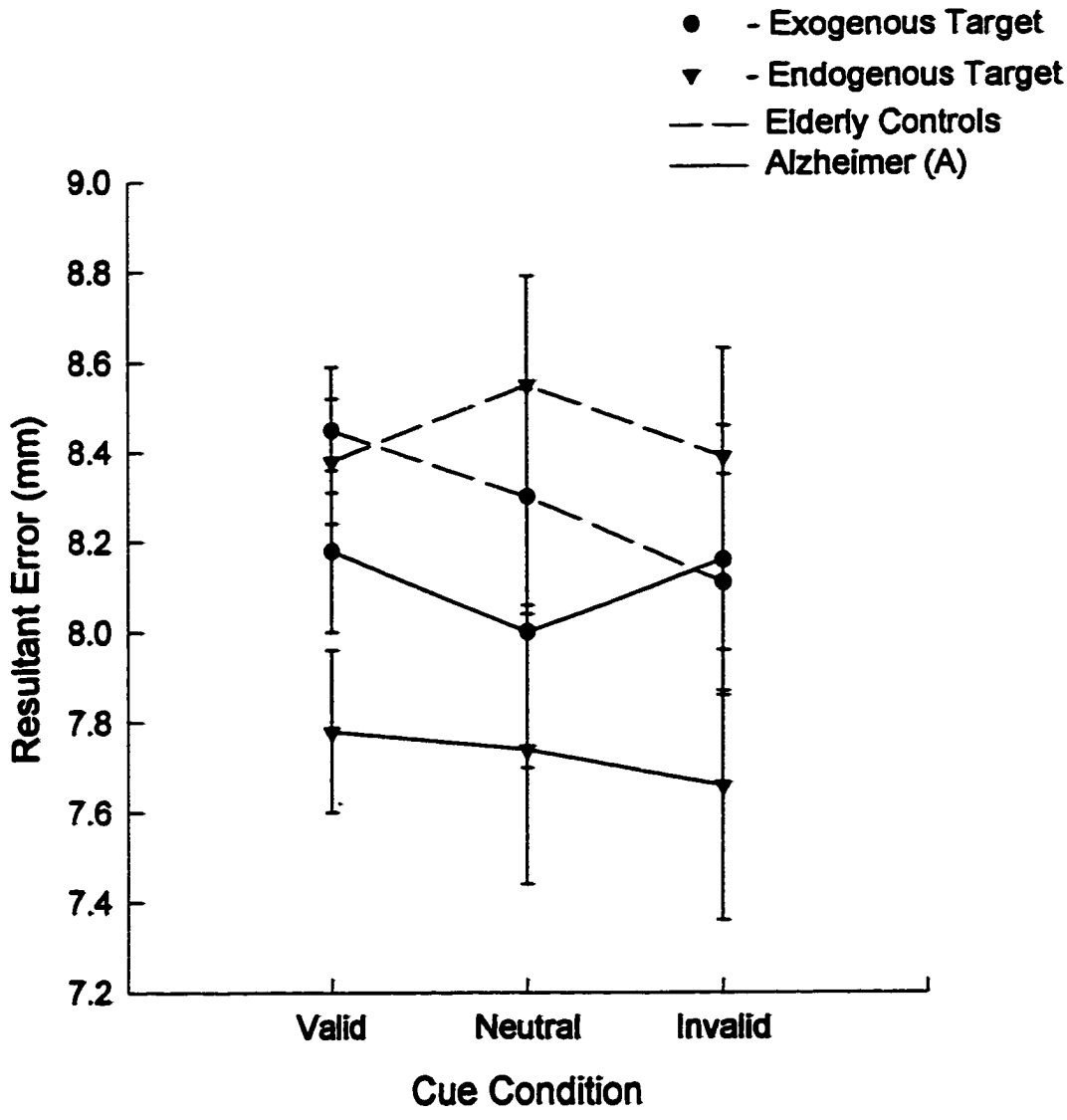


Figure 6.10 - Resultant error as a function of cue condition, target paradigm and group. The error bars represent the standard error of the least squares means.

ANALYSIS 3

Analysis 3 was a 3 (Cue Condition - Valid, Neutral, Invalid) X 2 [Group - Alzheimer(A), Alzheimer(B)] repeated measures MANOVA using SAS General Linear Models procedures. Group was a between subject factor and cue condition was a within subject factor. Target type could not be included as a variable since the AD(B) group completed only the exogenous target paradigm. That is to say that this analysis is confined to the Exogenous Paradigm. Dependent variables were identical to those of analysis 1.

One must keep in mind that the sample size is small for the AD(B) group ($n = 4$), and that this group developed out of the inability of these subjects to perform the Endogenous Target Paradigm. Thus, the power of this analysis and the generalizability of the results will be, necessarily, limited.

RESULTS

The MANOVA revealed multivariate main effects of group, $F(4, 9) = 5.13$, $p < .02$, and of cue condition, $F(8, 44) = 4.29$, $p < .0005$. There was no multivariate group by cue condition interaction, $F(8, 44) = 1.38$, $p = .23$. The significance of the multivariate analysis for group and cue condition allowed the inspection of the univariate mixed model Analysis of Variance (ANOVA) for each of the five dependent variables.

Reaction Time (See Figure 6.11)

Results of the univariate mixed model ANOVA for RT

revealed a main effect of group, $F(1, 12) = 13.76$, $p < .005$. Comparing least squares adjusted means (collapsing across target type) indicated that RT was an average of 671.3ms faster for the AD(A) group than the AD(B) group ($t = 3.19$, $p < .01$). There was a significant main effect of cue condition, $F(2,29) = 14.6$, $p < .0001$. Comparison of the least squares adjusted means (collapsing across target type and group) indicated that RT for the invalid and neutral conditions did not differ ($M = 1229.9$, $SE = 62.8$ms and $M = 1253.5$, $SE = 61.7$ms respectively; $t = 0.26$, $p = .79$) while the valid condition had a faster RT ($M = 967.5$ms, $SE = 36.9$ms) than the invalid ($t = 3.60$, $p < .001$) or the neutral conditions ($t = 3.97$, $p < .001$). There was no group by condition interaction, $F(2, 30) = .23$, $p = .80$ indicating that the pattern of results was equivalent for the two groups (as were the "costs plus benefits").

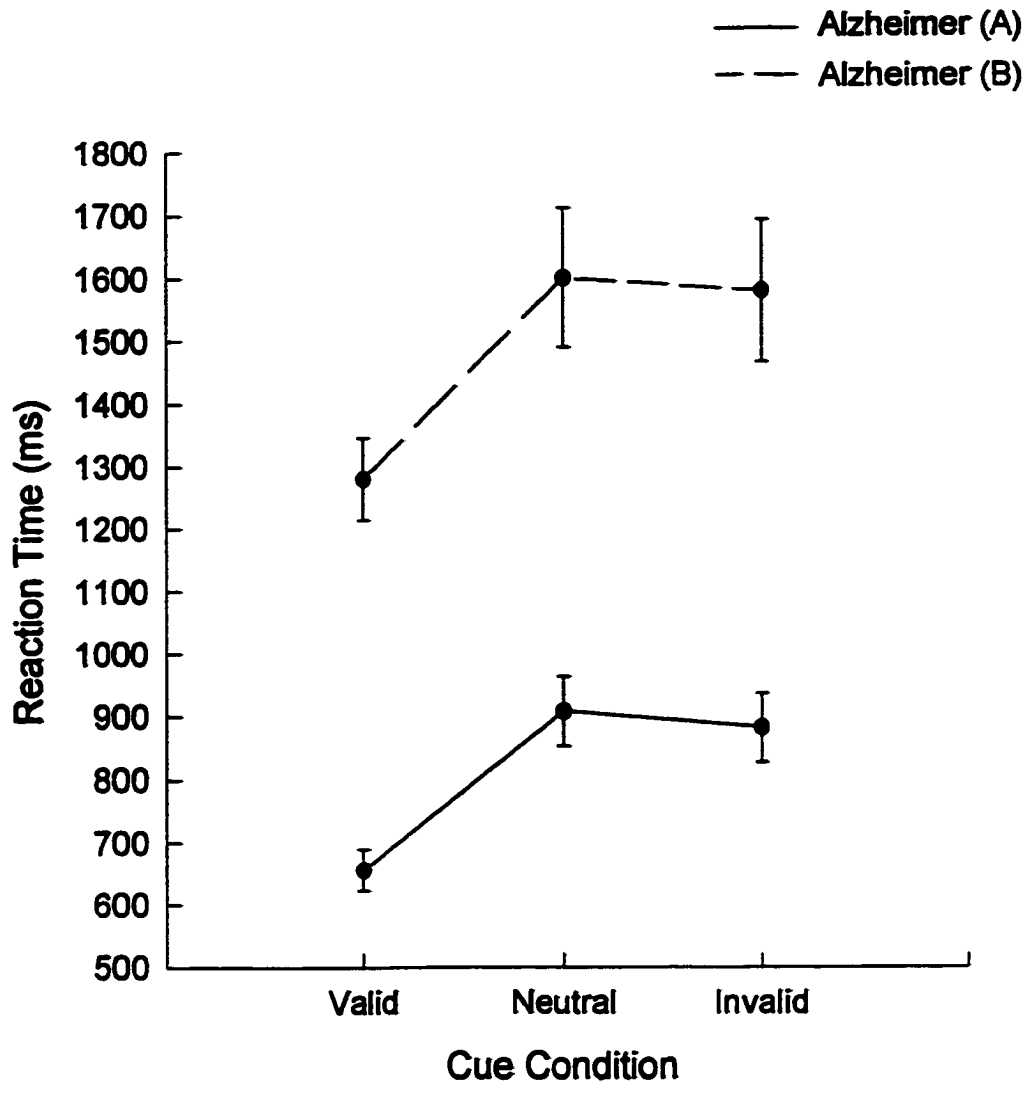


Figure 6.11 - Reaction time as a function of cue condition and group. The error bars represent the standard error of the least squares means.

Movement Duration Time (See Figure 6.12)

Results of the univariate mixed model ANOVA for MT revealed a main effect of group, $F(1, 12) = 8.12$, $p < .02$. Comparing least squares adjusted means (collapsing across cue condition) indicated than MT was an average of 140.5ms faster for the AD(A) group than the AD(B) group ($t = 2.45$, $p < .05$). There was a significant main effect of cue condition, $F(2,36) = 13.4$, $p < .0001$. Examination of the least squares adjusted means revealed that the MT was longest for the invalid condition ($M = 882.2$ms, $SE = 12.2$ms), followed by the neutral condition ($M = 837.3$ms, $SE = 11.9$ms) and the valid condition ($M = 811.7$ms, $SE = 7.2$ms). The invalid and the neutral conditions differed significantly ($t = 2.63$, $p < .05$) as did the invalid and valid conditions ($t = 4.99$, $p < .0001$). The neutral and the valid conditions differed only marginally ($t = 1.83$, $p = .08$).

There was a group by condition interaction, $F(2, 38) = 7.02$, $p < .005$ which reflected the fact that there were no differences in MT as a function of cue validity for the AD(A) group (all p values > .05). However, for the AD(B) group, MT was longer for the invalid condition than for either the valid ($t = 4.77$, $p < .0001$) or the neutral conditions ($t = 2.62$, $p < .05$). Valid and neutral conditions were equivalent for the AD(B) group ($t = 1.65$, $p = .11$) while the "costs plus benefits" was 121.4ms ($t = 4.77$, $p < .0001$).

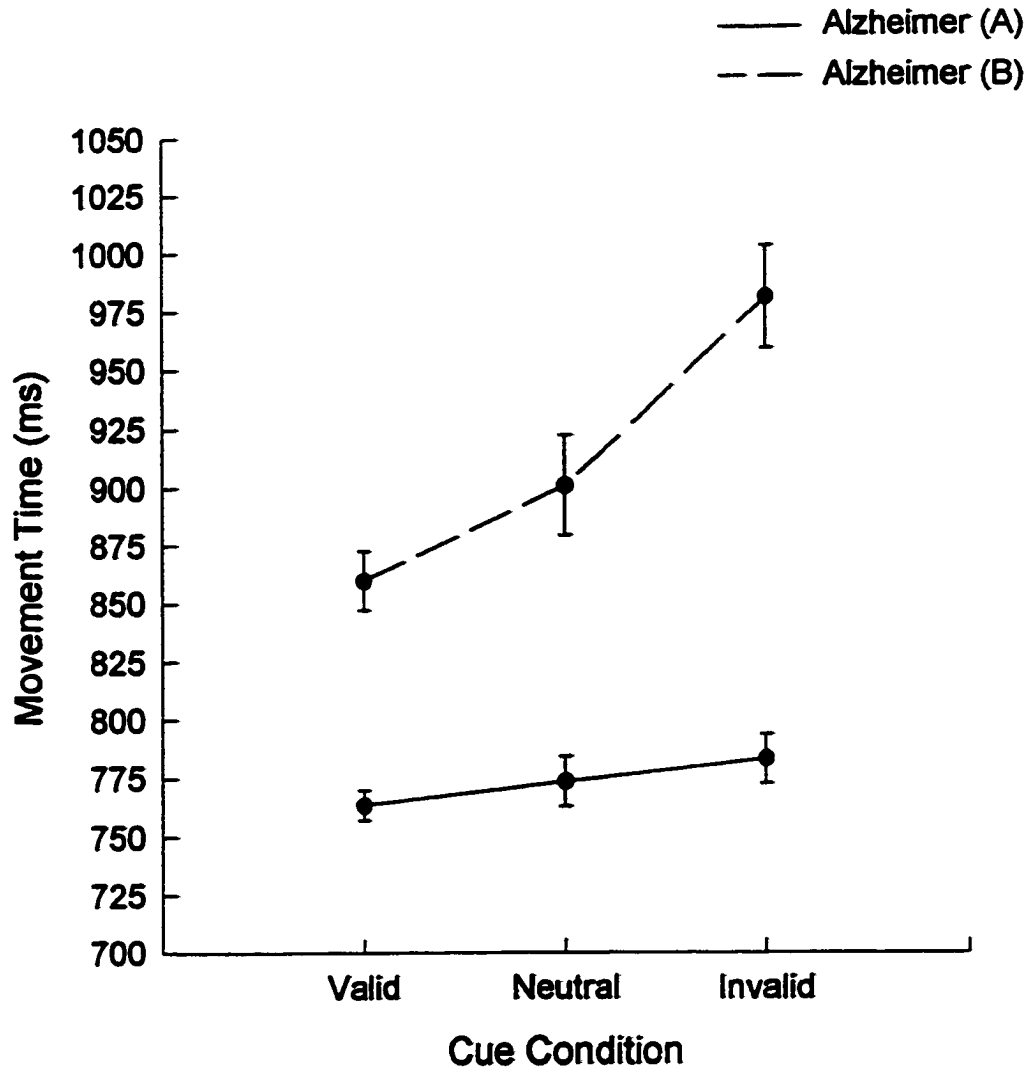


Figure 6.12 - Movement time as a function of cue condition and group. The error bars represent the standard error of the least squares means.

Peak Velocity (See Figure 6.13)

Results of the univariate mixed model ANOVA for PV revealed a main effect of group, $F(1, 12) = 5.96$, $p < .05$. Comparison of least squares adjusted means indicated than PV was an average of 12.9cm/sec higher for the AD(A) group than the AD(B) group ($t = 2.09$, $p = .05$). There was no significant main effect of cue condition, $F(2,36) = .37$, $p = .69$. There was no group by condition interaction, $F(2, 38) = 0.35$, $p = .71$, indicating the slightly lower PV for invalid target responses by the AD(B) group was not of statistical significance.

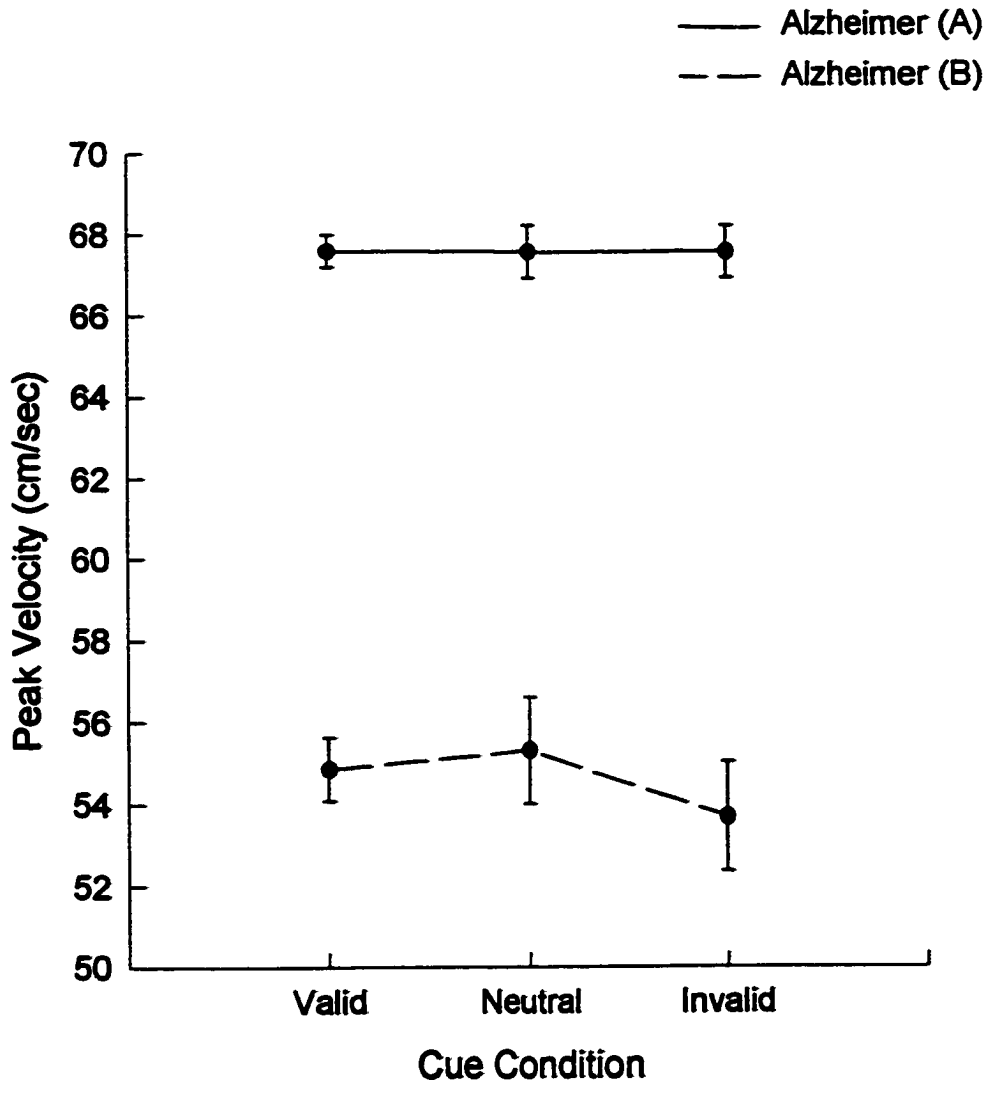


Figure 6.13 - Peak velocity as a function of cue condition and group. The error bars represent the standard error of the least squares means.

Percent Deceleration (See Figure 6.14)

Results of the univariate mixed model ANOVA for percent deceleration revealed no main effect of group, $F(1, 12) = 2.43$, $p = .14$, no main effect of cue condition, $F(2,31) = 1.33$, $p = .27$, and no group by condition interaction, $F(2, 32) = 0.22$, $p = .80$.

Resultant Error (See Figure 6.15)

Results of the univariate mixed model ANOVA for resultant error revealed no main effect of group, $F(1, 12) = .02$, $p = 0.88$, no main effect of cue condition, $F(2,74) = 1.49$, $p = .23$, and no group by condition interaction, $F(2, 80) = 1.79$, $p = .17$.

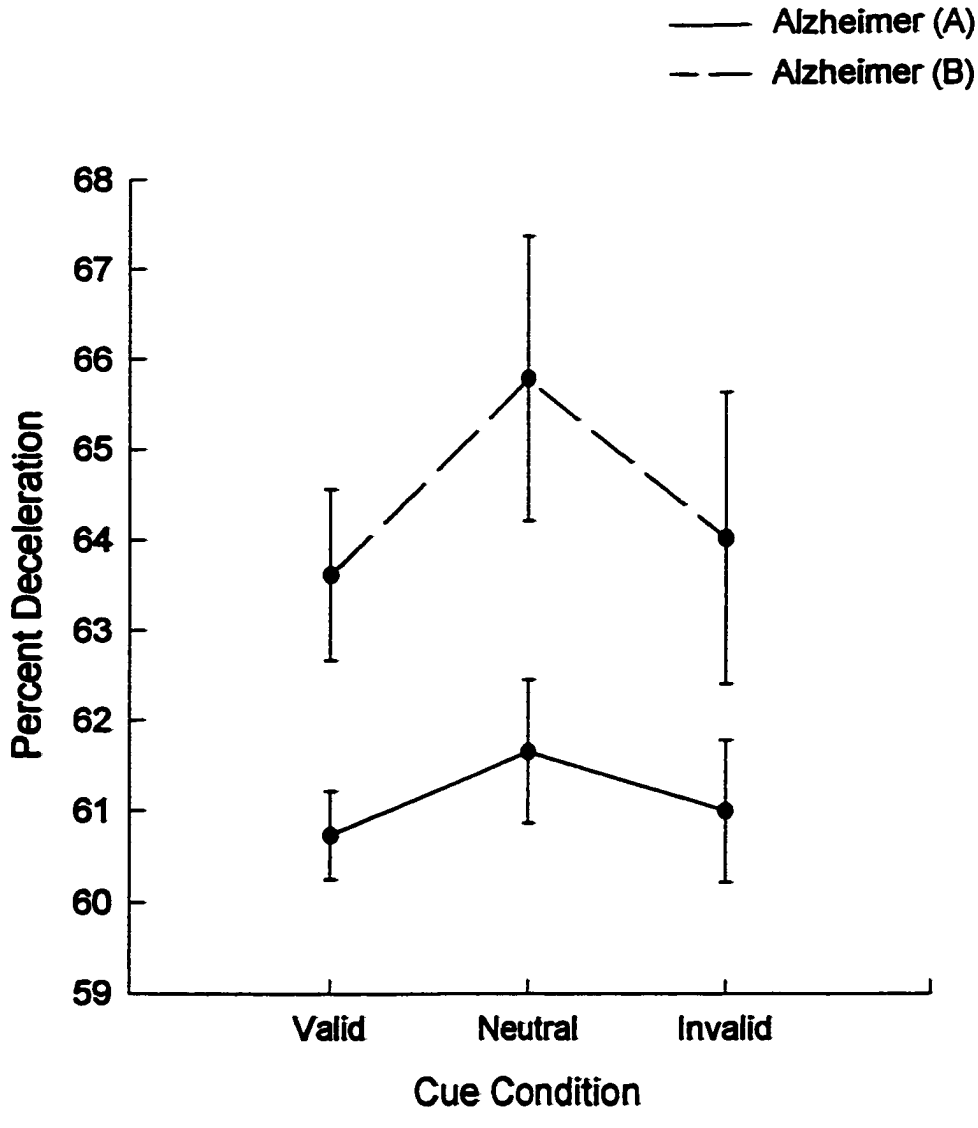


Figure 6.14 - Percent deceleration as a function of cue condition and group. The error bars represent the standard error of the least squares means.

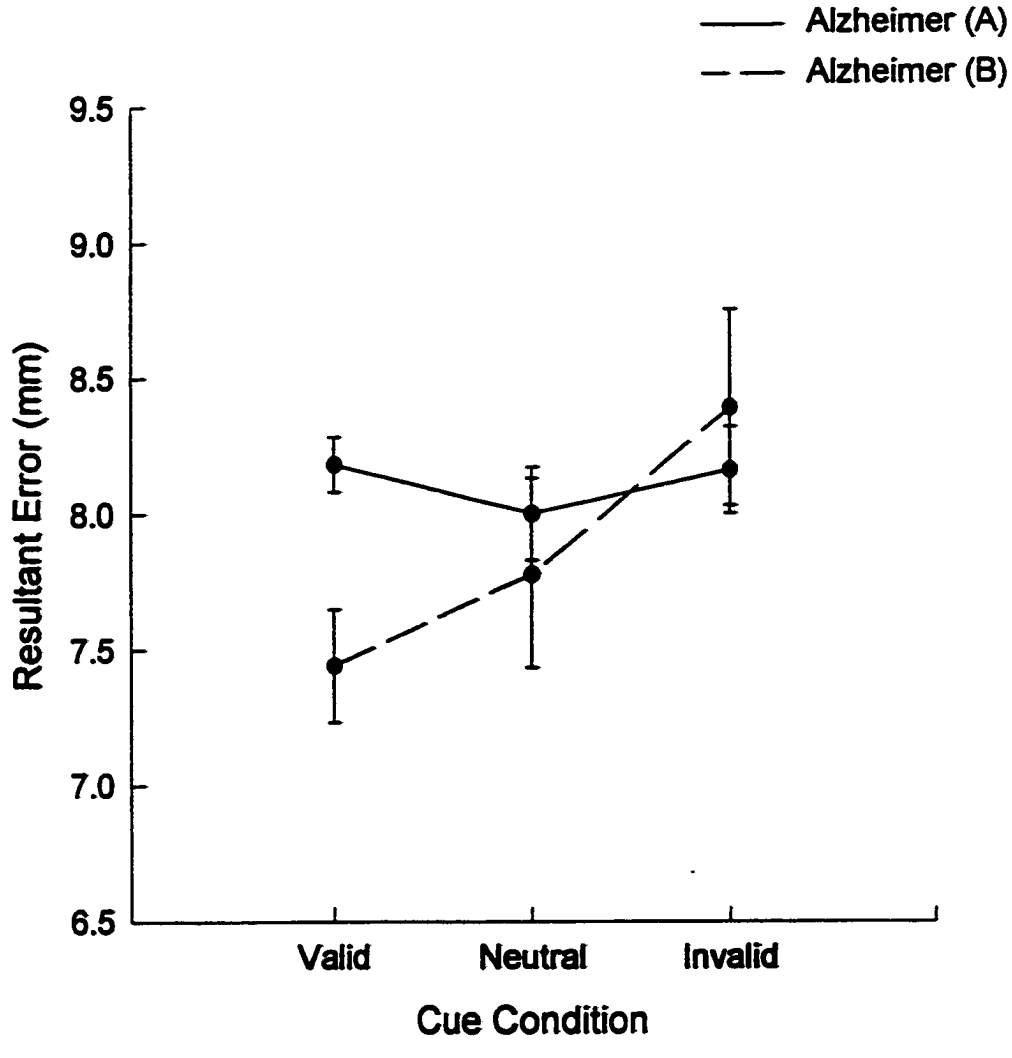


Figure 6.15 - Resultant error as a function of cue condition and group. The error bars represent the standard error of the least squares means.

Total Performance Time (RT plus MT) (See Figure 6.16)

Fisk and Goodale (1988) conducted a study in which subjects with either right or left hemisphere pathology performed visually directed pointing movements. They found that while the right hemisphere group took longer to initiate a movement than controls, they had equivalent MT to controls. In contrast, the left hemisphere group had equivalent RT to controls but exhibited a longer MT. Thus, despite obvious differences in performance, these two patient groups did not differ in the total time (i.e., RT plus MT) that they required to perform the tasks. Therefore it is of interest from an illustrative standpoint to examine the combined changes in RT and MT in the groups used in this study.

Figure 6.16 illustrates that for the Exogenous Target Paradigm, movements to invalidly and neutrally cued targets (i.e., unexpected events) took approximately 24% longer for the AD(A) group to complete that the elderly control group while movements to validly cued targets took approximately 15% longer for the AD(A) group than for elderly controls. For the AD(B) group, total performance time for all cue conditions was approximately 50% longer than that of the AD(A) group.

For the Endogenous Target Paradigm, movements to invalidly and neutrally cued targets took approximately 46% longer for the AD(A) subject group than for the elderly controls while movements to validly cued targets took approximately 30% longer. The AD(B) group did not complete the

endogenous target paradigm and thus could not be compared to the elderly controls.

As was noted in the previous comparison of healthy young and healthy elderly subjects, the greatest proportion of increase in the total time required to complete the task for the AD groups compared with the elderly control group and the AD(A) and AD(B) groups (in the Exogenous Paradigm) was the increase in RT rather than MT.

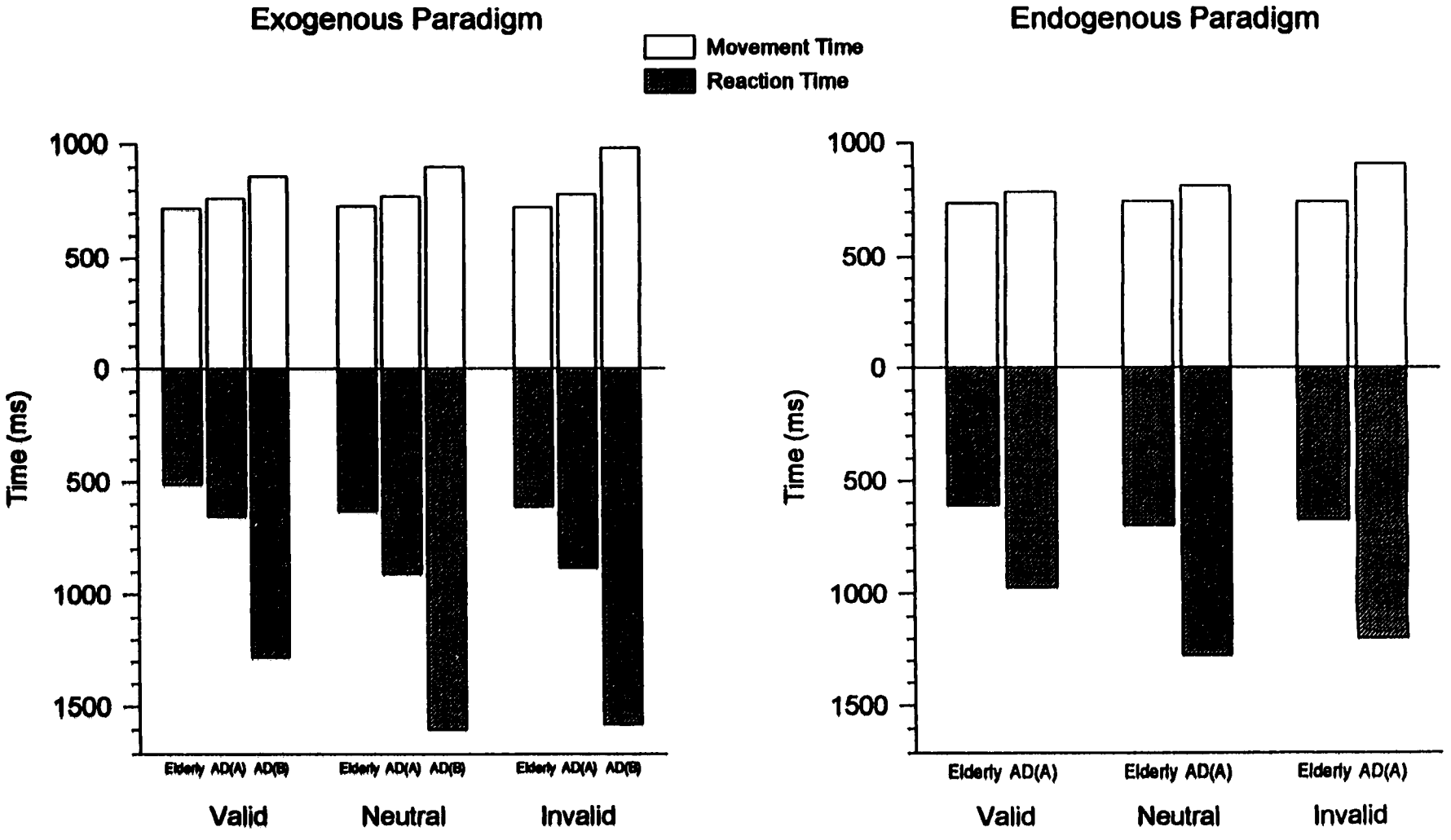


Figure 6.16 - Total Performance Time

DISCUSSION

The AD(A) group had, overall, faster RT and shorter MT for the Exogenous (compared with the Endogenous) Paradigm. Examining the "costs plus benefits" revealed only a RT advantage for the Exogenous Paradigm. In contrast, for the Endogenous Paradigm, there was a RT, a MT, and a PV advantage for valid cues. The MT and PV advantage was relative to the great disadvantage of these measures for the invalid cue conditions of the Endogenous Paradigm (refer to Figures 6.2 and 6.3).

Subjects in all three groups (AD[A], AD[B], elderly controls) were able to initiate movements faster to validly than to invalidly cued targets. This indicates that all groups were able to use advance information about a probable target location to facilitate the initiation of a movement. This advantage was limited to exogenous overt movements for the AD(B) subject group, however, since they were not able to perform appropriately in the Endogenous Paradigm.

The lack of any differences in accuracy among the three groups suggested, once again, that all subjects set accuracy as the primary goal of movements. While other studies of attention have reported that AD subjects were less accurate than healthy elderly subjects, such studies have used different measures of accuracy such as errors in letter discrimination (e.g., Parasuraman et al., 1992) or procedural errors (e.g., moved eyes too soon from fixation, moved eyes in

the wrong direction) when making saccades to visual targets (Scinto et al., 1994). In contrast, the current study found that when AD subjects were provided time to make visually directed movements, they were able to make corrections to a movement program either prior to initiation or during execution in order to achieve equivalent accuracy to elderly controls.

The deficiencies in performance of the AD group were only evident when measures other than accuracy were examined. The AD(A) group were slower, overall, to initiate movements (by approximately 357ms), took longer to complete the movements (by approximately 72ms), and reached a lower average PV (by approximately 7cm/sec) compared with elderly controls. These differences in RT are consistent with the results of previous research which has examined RT differences in aging and in AD (e.g., Bellgrove et al., 1997; Madden, Nebes, & Allen, 1992; Pate et al., 1994). With the exception of Bellgrove et al. (1997), MT and PV have not previously been examined in the motor programming of aiming movements in AD research. The MT and PV differences in the current study were much less than those found in Bellgrove et al.'s (1997) study. This is likely accounted for, in part, by the relatively simple, discrete single movement used in the current study compared with the more complex, four-step sequential movement used by Bellgrove et al. (1997). Level of cognitive functioning was also a possible contributor to these differences as the subjects used

by Bellgrove et al. (1997) were more cognitively impaired than those used in the current study (i.e., one-third of the 12 subjects in their study had MMSE scores below the range of scores for the subjects in this study). Bellgrove et al. (1997) did note that five of their subjects were unable to complete the "no cue" condition but the cognitive status of these subjects was not described further. In the present study, the more impaired subjects [AD(B)] were slower than elderly controls to initiate movements (by approximately 670ms), took longer to complete the movement (by approximately 140ms), and reached a lower average PV (by approximately 13cm/sec) in the Exogenous Paradigm. Thus, the greater decline of the AD(B) group's performance is consistent with the suggestion that the performance deficits (e.g., slowing of RT) become greater in AD as the level of cognitive functioning decreases (Nebes & Brady, 1992).

Examining the contribution of RT and MT to total performance time suggested that the majority of the differences among the three cue conditions was due to proportionately greater increases in RT compared with MT. This was true of both the healthy elderly subjects and the AD subjects and, once again, suggests that the majority of cognitive processing and motor programming were conducted during the RT interval. The generalized slowing of these processes that was evident in the comparison of healthy young and elderly subjects increased dramatically with AD and became

more pronounced as the level of cognitive ability (in AD) declined. Nestor, Parasuraman, and Haxby (1991), have attributed such slowing of information processing in AD to deficits in executive functioning of the frontal lobes.

Although memory deficits are the hallmark of AD (Carlesimo & Oscar-Berman, 1992) the current study required procedural learning rather than retrieval from episodic or semantic memory, the characteristic memory deficit of AD. The experimenter also prompted the subjects on the sequence of instructions prior to each trial. Furthermore, in the Exogenous Paradigm the target "automatically" appeared in the periphery and in both the Exogenous and Endogenous Paradigms the cue remained on until the target was presented, thereby reducing any memory demands. For the Endogenous Paradigm, subjects did have to remember where the target numbers were on the screen (i.e., target 1 and 2 were left of center and target 3 and 4 were right of center) in order that they would not have to scan the screen for each trial but merely look to the target location and point to it. However, the positions of the numbers were constant throughout the two paradigms (1 to 4, left to right) and ample practise was provided. Thus, memory problems, per se, were unlikely to have contributed to AD subjects impaired performance on these tasks.

RT was faster for the Exogenous Paradigm than for the Endogenous Paradigm for both the AD(A) and the elderly control groups, indicating that the advantage of exogenous over

endogenous overt movements that was noted in the healthy control groups was not affected by the pathology associated with mild AD (i.e., AD, group A) subjects. Thus, the AD(A) group (i.e., mild AD patients) appeared to be able to use advance information to preprogram motor acts. However, the performance of the mild AD group did reveal deficits in their ability to use advance information efficiently. The differences in RT between the Exogenous and Endogenous Paradigms were much greater for the AD(A) group (334ms) compared with the elderly control group (77ms) and, unlike the elderly control group, the exogenous target advantage (or the endogenous target disadvantage) extended to both MT and PV measures. Thus, overall, the AD(A) group were much more slowed in the Endogenous Paradigm than in the Exogenous Paradigm relative to healthy controls. This discrepancy suggests that the AD subjects were more dependent on external information (such as that provided in the Exogenous Paradigm) for the planning and execution of movements than were elderly controls. The greater RT differences between the Endogenous and Exogenous Paradigms for the AD subjects compared with the elderly control subjects may have been due to a greater time required for the AD subjects to decode the meaning of the numeric target cue.

The elderly control and the AD(A) groups had a similar pattern of results for the Exogenous Paradigm in that RT was the only variable affected by cue validity. This suggests

that, for exogenous overt movements, both elderly controls and individuals with mild AD (i.e., AD group A) completed all necessary reprogramming, of a previously incorrectly planned movement, prior to movement initiation. However, the RT advantage of the valid cue and the RT disadvantage of the invalid target (i.e., "costs plus benefits") was much greater for the AD(A) group than for the elderly control subjects. This indicates a general slowing in the processes involved in shifting attention and/or reprogramming a movement, for mild AD subjects.

For the Endogenous Paradigm, a different pattern of results emerged for the mild AD group and this "qualitative" difference in performance of the task suggested more than just a slowing of information processing for this paradigm. In addition to the "costs plus benefits" in RT that was noted for the elderly controls, the AD(A) group also had longer MT and lower PV for invalidly cued compared with validly cued targets. Thus, in contrast to the elderly controls, the AD(A) subjects appeared to complete only part of the necessary reprogramming for endogenous overt movements prior to movement initiation and made additional adjustments during movement execution in order to attain adequate accuracy. Since they did not demonstrate an increase in the percent deceleration for the invalid cue condition, adjustments to the movement trajectory did not appear to be confined to terminal corrections during the deceleration phase but also appeared to

occur during the acceleration phase of the movement.

The "costs plus benefits" for the AD(A) group for both paradigms was significantly greater than the "costs plus benefits" for healthy elderly for both paradigms (101.5ms and 63.4ms). Thus, even the mild AD group was impaired relative to the elderly control group. The AD(A) group had equivalent RT "costs plus benefits" for the Exogenous and Endogenous Paradigms (226.2ms vs 225.3ms, respectively). This suggests the possibility that the mild AD subjects may not have shifted their attention in response to the cues in the Exogenous Paradigm but merely prepared a motor response (see Alternative #3, pg. 49). Alternatively, the AD subjects may not have either shifted attention or prepared a movement in either of the Paradigms (see Alternatives #7 pg. 51 and Alternative #6, pg. 54). Since neutral and invalid cues had equivalent RT for the AD group in the Endogenous Paradigm, once again, the neutral cue did not provide an appropriate baseline to examine "costs" and "benefits" separately (e.g., Jonides & Mack, 1984).

It is somewhat ironic that the pattern of results of the AD(A) subjects in the Endogenous Paradigm was actually similar to that of young healthy adults in that both groups made some changes to an incorrect preprogrammed movement prior to its initiation and made further adjustments during movement execution. However, while the patterns are similar, the AD group was much less efficient in conducting their movements

than the healthy young adults as indicated by their dramatically longer RT and MT as well as their lower PV. Thus, while this strategy allowed the young subjects to maintain their efficiency in producing accurate movements, for the AD subjects, it appeared that there was an inability to completely respecify the motor program for an endogenous target such that additional adjustments were required in order to attain adequate accuracy.

That the mild AD were impaired relative to healthy elderly controls (slower RT, longer MT, lower PV) suggests that the mild AD group had particular difficulty with unexpected changes in the position where they expected the target to be located. This was particularly problematic if they had to make changes to a movement without having an external event to pull their attention to the new, correct target position (i.e., endogenous vs exogenous paradigms).

AD(A) Group Versus AD(B) Groups

The AD(B) group had a very small sample size (n = 4). This sample was not chosen by the experimenter, but rather, was based on their inability to perform the Endogenous Paradigm. Thus, the statistical power of this analysis and its generalizability is limited.

While the AD(B) group was slower overall than the AD(A) group, the RT "costs plus benefits" did not differ significantly between the AD(A) and the AD(B) groups in the Exogenous Target Paradigm. Thus, the more advanced AD subject

group did not appear to be more impaired than the mild AD group on preparation of a response during the RT period. However, in contrast to the AD(A) group, who appeared to make all corrections to an incorrectly preplanned exogenous movement prior movement initiation, the AD(B) group appeared to require additional adjustments during the execution of the movement. The significant "costs plus benefits" in MT for the AD(B) group (121.4ms), and the absence of such differences for the AD(A) group suggests that overall, the moderately impaired AD subjects were less able to complete the necessary reprogramming of the movements in the RT interval.

It is noteworthy that the MT "costs plus benefits" in the Exogenous Target Paradigm for the AD(B) group were equivalent to those of the AD(A) group in the Endogenous Target Paradigm (121.4ms). This suggests that the moderately impaired subjects' deficits in reprogramming exogenous overt movements was similar to the mildly impaired subjects' reprogramming deficit for endogenous overt movements. These results suggest the possibility that there is a hierarchy of decline in ability when motor programming is combined with exogenous and endogenous overt movements. In mild stages of the disease, it appeared that the ability to reprogram an exogenous overt movement was intact, although additional time was required for the processes of programming the movement. However, when there was a requirement to reprogram an endogenous overt movement, mildly impaired AD subjects were unable to completely

respecify the motor program requirements and had to do so during the execution of the movement. In contrast, those with more severe cognitive deficits were unable to produce endogenous movements and were unable to respecify this motor program for exogenous overt movements. It seems likely that the more cognitively impaired AD subjects required the presence of exogenous targets in order to reprogram their movements. Another manner in which to conceptualize this possible hierarchy of decline in AD would be from the perspective of "automatic" and "controlled" processing. Automatic processing is described as a "fast, generally parallel, fairly effortless process...that is not under subject control", while controlled processing is described as "a slow, generally serial, effortful, subject regulated processing mode" (Schneider, Dumais, & Shiffrin, 1984, p. 1-2). Exogenous cues and exogenously presented targets (i.e., abrupt onset targets) have been described as eliciting and "automatic" orienting of attention (e.g., Jonides & Yantis, 1988). In contrast, endogenous cues and, presumably, endogenously presented target information, have been described as being under the cognitive control of the individual who makes a voluntary decision to shift attention (e.g., Klein et al., 1992).

The Exogenous Paradigm used in the current thesis may be considered to be analogous to "automatic processing" in that it has an abrupt onset target (known to draw attention

automatically and not to require cognitive resources), while the Endogenous Paradigm may be considered analogous to "controlled processing" (in that the target was presented centrally, requiring the subject to make a voluntary decision using cognitive resources). Thus, while mildly impaired AD subjects [i.e., AD(A)] were most obviously impaired on a controlled processing task (i.e., Endogenous Paradigm), AD subjects with more advanced impairments [i.e., AD(B)] were completely unable to perform the controlled processing task and had obvious impairments on an automatic processing task (i.e., Exogenous Paradigm). This suggests that there may be a hierarchy of decline in AD whereby controlled (endogenous) processes are the first to decline, and, as the disease progresses, the automatic (exogenous) processes also decline. Further, by the time the automatic processes begin to decline, the controlled processes are severely impaired. Such findings support the opinion of Jorm (1986), who, in a review of the literature, concluded that controlled processing is affected early in the AD process and that automatic processing remains intact until later in the disease, at which time it, too, deteriorates. Similarly, Brodeur and Enns (1997), in conducting a life span analysis of exogenous and endogenous orienting, concluded that automatic processing remains intact longer in the life span than voluntary processing. From their review of the literature, they concluded that exogenous orienting involves the mid-brain structures (superior

colliculus, thalamus) in cooperation with the parietal lobe. In contrast, they concluded that endogenous orienting involves the cooperation of the parietal and pre-frontal cortex. Brodeur and Enns (1997) point out that human brain development proceeds very quickly from the midbrain structures and more slowly for the primary cortical areas (e.g., parietal lobes) and slowest for the prefrontal cortex. These conclusions led them to postulate that automatic processing develops earlier and remains intact longer than voluntary processing (which develops later and deteriorates first). They labelled this the "first in, last out" hypothesis. The present results extend Brodeur and Enns' (1997) conclusions regarding normal aging to stages of pathology in AD.

Although most of the literature regarding attentional deficits in AD have focused on posterior (i.e., parietal) cortex functioning, frontal lobe pathology has been associated with deficits in planning and executing movements, in attention and concentration, and in the ability to monitor ones thoughts and actions (Rhawn, 1990), all of which are important in the current paradigm. Sakata, Taira, Mine, and Murata (1992) proposed a model of interactive communication between the frontal and parietal systems for visually guided hand movements. This model is consistent with other research which has found that both the frontal and parietal lobes are involved in visual attention and motor function (e.g., Andersen, 1989, 1995; Grafton et al., 1992; Lamarre & Chapman,

1986; Requin, 1992). Sakata et al. (1992) suggest that visual information is first processed in the posterior parietal lobe and this visual information is sent to the premotor cortex where a hand movement is programmed. A copy of this program is sent back to the visual and motor neurons in the parietal lobe and, if the program matches the visual signal (which would be the case in a valid cue condition), the parietal cells send a signal back to the premotor cortex instructing it to execute the movement. If the program and the visual signal do not match (which would be the case in a invalid cue condition), an inhibitory signal is sent back to these visual and motor cells instructing them to interrupt or modify the movement. According to this model, the parietal cortex also monitors hand movements and the match between the movement program and the visual target. Thus, given that both parietal and frontal pathology has been documented in AD, it is not surprising that this disease process may affect visually directed reaching movements. The model proposed by Sakata et al. (1992) could be used to account for the longer RT noted in the AD subjects (for those processes that are conducted prior to movement initiation) and for the longer MT (presumably from online corrections through monitoring). As the severity of neuropathology increases, these processes may be further affected which would support the longer RT and MT of the AD(B) compared to the AD(A) in the current study.

If the interaction between the frontal and parietal lobes

is responsible for the deficits noted in AD, the hierarchy of decline suggested by results of the current study fit well with the documented pattern of neural degeneration of frontal and parietal that accompany progression in AD. Parks, Haxby, and Grady (1993), in a review, pointed out that the cerebral metabolic rates for glucose in the frontal lobes (in mild AD) are reduced by 15% to 21%, and for the parietal lobes are reduced by 23% to 39%. As the disease progresses (to severe AD) the reduction in the frontal lobes increases to between 26% to 46% and is reduced in the parietal lobes by 40% to 50%.

Qualitative observations were also instructive in illustrating how the behavioral deficits of the AD subjects were manifest. AD subjects were noted to be unable to initiate a movement on occasion (e.g., pressed up and down on the computer mouse trigger but failed to lift finger off the trigger); often attempted to use the wrong finger, more than one finger, or the wrong hand when pointing to targets (more evident in the AD(B) group); and had a tendency, in the endogenous target paradigm, to point to the central target indicator rather than to the actual peripheral targets. One subject had insight into this problem and made the comment after being unable to compete a trial correctly "That's not right. Why do I get so confused?". This latter behavior may have been an example of exogenous (automatic) orienting of attention overriding endogenous (controlled) attempts to shift attention as the central target information may have been

processed as an abrupt onset target. Deficits in the ability to inhibit an inappropriate response have been described by Stuss and colleagues in association with deficient frontal system functioning. One subject from the AD(B) group often would press down on the mouse key numerous times but was unable to lift his finger to carry out the aiming task. This seems likely an example of motor perseveration, which is associated with frontal lobe dysfunction (Chatterjee, 1998).

For the Endogenous Paradigm, some AD subjects would say the number aloud, even when instructed that this was not necessary. This verbalization may have been an example of a failure of response inhibition and appeared to interfere with the ability to make an actual movement with their finger (i.e., they either did not move their finger or moved it only after they finished verbalizing). In this situation, the task may have become a dual motor task (which is known to be associated with a decrement in one of the two motor tasks, which in this case was the finger movement). This problem was most noticeable for one of the subjects in the AD(B) group.

This type of response also appears to be somewhat consistent with apraxia, in that the subject had difficulty learning the sequence of the task, although apraxia is not usually noted in those AD patients with such mild impairments. Another explanation that has merit is that of LaFleche and Albert (1995) who suggested that AD subjects have the greatest difficulty on tasks of executive function that require the

concurrent manipulation of information (even for tasks where the individual aspects of the task can be conducted). The Endogenous Paradigm had a much greater demand for the manipulation of concurrent information than did the Exogenous Paradigm.

AD subjects also had a tendency not to take their hand down from the target on the screen after completing the movement, would not place their finger back on the mouse trigger key, and would forget to depress the trigger key to initiate a new trial after placing their finger on it. This may suggest facilitory paratonia (the inability to relax the limb and actively aid movements) which is associated with dysfunction of the frontal lobes (Beversdorf & Heilman 1998; Chatterjee, 1998).

It is important to keep in mind that, from a practical "everyday" perspective, these experiments involved simple movements, presumably, with a low cognitive load (for the average healthy person). The delay in RT noted by an unexpected event (i.e., invalid cue), in AD subjects (compared with healthy elderly), even when the target was exogenous has implications for many activities of daily living.

CONCLUSIONS

As was the case with the young and the healthy elderly subjects, accuracy was a priority for both AD groups. However, in order to maintain accuracy they required much longer times to initiate and complete the movements. AD subjects generally

had longer RT, slower MT, and lower PV compared with elderly controls and these effects became even more pronounced as cognitive ability deteriorated [i.e., AD(B)].

AD subjects with mild cognitive impairments [i.e., AD(A)] were able to use cues to preprogram both exogenous and endogenous overt movements when provided with endogenous premovement cues. However, when required to reprogram endogenous overt movements, even the mild AD group was impaired compared with elderly controls and the more severely cognitively impaired were unable to complete the task. The AD group did not appear to have shifted attention in response to the cues in the Exogenous Paradigm (as indicated by the equivalent "costs plus benefits") but merely prepared the motor response.

The advantage of the Exogenous over the Endogenous Paradigm that was noted for the healthy elderly subjects was maintained in the AD subjects. For the AD(A) group this facilitation was confined to RT while for the AD(B) group this was also reflected in MT and PV. This suggests that the AD(A) subject group had deficits in visuomotor integration for exogenous overt movements in comparison with the healthy elderly and that, with disease progression [AD(B) group], these deficits may become much more pronounced (i.e., longer RT, longer MT, lower PV). Further, these results suggest that the AD(A) subjects (similar to the healthy elderly) made all changes to an incorrectly pre-programmed movement prior to

movement initiation for exogenous overt movements. For endogenous overt movements, the AD(A) subjects made further adjustments during movement execution (in contrast to the healthy elderly subjects). The AD(B) group had a similar pattern for the Exogenous Paradigm as the AD(A) group for the Endogenous Paradigm indicating that they completed some programming prior to movement initiation but also required adjustments to be made during movement execution. These results suggest the possibility that there may be a hierarchy of decline in AD whereby motor programming of endogenous overt movements (endogenous or controlled processing) shows deficits early in the disease and that motor programming of exogenous overt movements (exogenous or automatic processing) deteriorates with disease progression (at which time endogenous processing is severely deteriorated).

The deficits revealed for the AD subjects (and the hierarchy of decline based on level of cognitive deficits) in the current study seem best accounted for by disruptions in the integration of communication between the frontal and parietal lobes due to progressive worsening of the pathology to both areas with disease progression.

The current chapter examined covert shifts of visual attention and motor programming in AD - a disease considered to affect primarily cognitive processes. The following chapter will examine these issues in Parkinson's Disease (PD) which is considered to affect primarily motor processes.

Chapter 7 - Study 4

THE EFFECTS OF VISUAL ATTENTION AND MOTOR PROGRAMMING ON MANUAL AIMING MOVEMENTS IN INDIVIDUALS WITH PARKINSON'S DISEASE (PD)

INTRODUCTION

Parkinson's Disease (PD) is a chronic, progressive disorder of the central nervous system first described by James Parkinson in 1817. Symptoms include akinesia (deficits in movement initiation), bradykinesia (slowness of voluntary movement), hypokinesia (reduction in movement amplitude and speed), muscle rigidity, and tremor of the hands and wrists (particularly at rest), and, to a lesser degree the head and neck (Playfer, 1989). Disturbances of posture and gait, and lack of facial expression are also characteristic (Stacy & Jankovic 1992) as are micrographia, loss of righting reflexes, and reduced rate of eye blink (Bradshaw & Mattingley, 1995). Cognitive deficits are also associated with PD, although these have received relatively little attention until recent years (for a review see Fisk & Doble, 1992). These include deficits in memory, spatial, conceptual, and attentional abilities, and bradyphrenia (slowness of thinking/information processing). Estimates of the prevalence of dementia vary; however, most estimates are in the range of 20% to 40% of those individuals with PD. Mood disorders, particularly depression, have also been noted to occur in PD with both reactive and endogenous etiologies proposed (Fisk & Doble, 1992).

PD has been estimated to be the third most common neurological disease in the elderly (Mahurin, Feher, Nance, Levy, & Pirozzolo, 1993). The incidence of PD is low under the age of 40 but increases greatly after age 70. The average age of onset is estimated at between 59 to 62 years (Bradshaw & Mattingly, 1995). Given its gradual onset and progressive nature, there can often be a considerable period between symptom onset and diagnosis making it difficult to be precise regarding disease onset for many patients. The prevalence of PD has been estimated to be approximately 0.5% in the population of 50 year olds, 1.0% in the population from 60 to 85 years of age, and 2.5% for those over 85 years of age (Bradshaw & Mattingley, 1995).

The rate of progression of PD varies but the majority of individuals experience significant disability within 10 to 15 years of disease onset. It has been estimated that approximately one in four individuals with PD experience rapid progression and become severely disabled within 5 years of disease onset while others remain relatively unimpaired for up to 10 years or more (Hoehn & Yahr, 1967).

There have been four variants of PD described: 1) idiopathic (cause unknown); 2) PD induced by neuroleptic drugs and by the neurotoxin 1-methyl-4phenyl-1,2,3,6-tetra-hydropyridine (MPTP); 3) PD resulting from encephalitis or influenza with fever; and 4) PD arising from atherosclerosis. Idiopathic PD is the most common type and has been estimated

to account for approximately 86% of cases (Bayles, Kaszniak & Tomoeda, 1987). Although the cause of idiopathic PD is unknown, hypothesized etiologies include viruses, premature or accelerated aging, genetic transmission and neurotoxins (Playfer, 1989). It has been suggested that these factors are not mutually exclusive (Barbosa, Limongi, & Cummings, 1997). To maintain a homogenous group, the diagnosis of all subjects in the present study was idiopathic PD (without dementia).

Diagnosis of PD

The diagnosis of PD is made by clinical examination and is based on the presence of characteristics of the disease. A Severity Rating Scale was developed by Hoehn and Yahr (1967) which includes the following five stages: Stage 1 - Unilateral motor symptoms which result in very little functional impairment; Stage II - Bilateral or midline motor symptoms, without impairment of balance; Stage III - Impaired righting reflexes (e.g., difficulty maintaining steadiness during turns); some restriction of daily activities, but still capable of independent living; Stage IV - Severe disability (the individual is able to walk and stand without assistance but has significant difficulty with other activities); and Stage V - Individual is confined to bed or wheelchair unless assisted. Stages 1, II, and III are considered to represent minimal impairment while stages IV and V are considered to represent severe disability (Hoehn & Yahr, 1967).

Behavioral and Cognitive Aspects of PD

While traditionally, PD has been considered to be a disorder of movement, it has been associated with many cognitive deficits (Fisk & Doble, 1992). Visuospatial deficits have been commonly documented in PD patients (Boller, Passafiume, Keefe, Rogers, Morrow, & Kim, 1984; Cummings & Huber, 1992; Levin, Llabre, Ansley, Weiner, & Sanchez-Ramos, 1990). These have been suggested to occur in the absence of dementia, to deteriorate further as the Hoehn and Yahr stage progresses, and to contribute to difficulties in activities of daily living (Boller et al., 1984). Slowness of information processing (bradyphrenia) has been noted to occur in PD (Revonsuo, Portin, Koivikko, Rinne, and Rinne, 1993) while deficits in memory, abstract thought, and executive function (e.g., set shifting, set maintenance, mental planning) have also been reported in PD (see Cronin-Golomb, 1990 for review). Taylor and Saint-Cyr (1995) contend that it is the deficits of executive functioning (particularly adapting to novel situations) that cause the greatest difficulty for PD patients.

Treatment of PD

While there is no cure for PD, there are symptomatic treatments. Anticholinergic and dopamine simulating drugs are the most common treatments. These drugs do not stop disease progression, however, and may lose their effectiveness over time. When drugs become ineffective, surgical procedures may

be considered. These include pallidotomy (i.e., surgical removal of the globus pallidus of the brain), grafting of dopamine-producing adrenal tissue into the striatum, and transplanting fetal substantia nigra cells into the striatum of the patient (see Feldman, Meyer, & Quenzer, 1997 for a review). These methods remain controversial, however, since their effectiveness has not been established clearly.

Neuropathology and Neurochemistry of PD

In PD, there is a reduction of the dopaminergic cells in the substantia nigra pars compacta (an estimated reduction of 50% compared with young healthy adults) and a reduction in dopaminergic neurons in the midbrain. There is also a reduction of noradrenergic neurons in the locus coeruleus and the dorsal vagal nucleus, a reduction of serotonergic neurons in the dorsal raphe nucleus, and a reduction of cholinergic neurons in the nucleus basalis of Meynert and in the pedunculopontine nucleus and the dorsal nucleus of the vagus (Feldman et al., 1997; Cornford, Chang, & Miller, 1995). There is the formation of Lewy bodies (abnormal spherical cytoplasmic bodies) within the remaining neurons of the brain stem (White, Au, Durso, & Moss, 1992).

Related neurochemical changes in PD include a depletion of dopamine in the striatum. This depletion is slightly greater in the pathway to the putamen (reduction of approximately 85%) than in pathway to the caudate nucleus (reduction of approximately 75%) (Agid et al., 1993), globus

palladus, and the substantia nigra. There is also a reduction of dopamine in the neocortex (although less than in the striatum), the limbic system, the hypothalamus, and the retina. PD symptoms do not usually become apparent until the striatal dopamine levels are depleted by 70% to 80%. Norepinephrine has been noted to be depleted by 40% to 80% in the cortex, hippocampus, amygdala, thalamus, hypothalamus, nucleus accumbens, substantia nigra, and the spinal cord. Serotonin has been reported to be reduced by approximately 50% in the basal ganglia, cortex, hippocampus, and spinal cord. Choline acetyltransferase (the enzyme responsible for synthesizing acetylcholine) is reduced in the cortex and the hippocampus. Deficits in various neuropeptides have also been noted in PD (for a review see Feldman, Meyer, & Quenzer, 1997). Most of these studies have been post-mortem. For this reason they will typically reflect late-stage changes and may overestimate the neurochemical abnormalities in patients with earlier stages of the disease (such as those subjects used in the current study).

Motor Functioning in PD

The motor deficits associated with PD have been linked to impairments in motor programming, movement initiation, movement termination, the switching of motor plans, and the integration of complex goal-directed movements. The time needed to prepare a movement has been shown to increase with movement complexity, the accuracy required, and the time

required to make a decision on the response in comparison with age-matched control subjects (see Mahurin et al., 1993 for a review). Most studies have found that RT is slower for PD than control subjects (Brown, Jahanshahi, & Marsden 1993; Jahanshahi, Brown, & Marsden 1992); and MT longer (Brown et al., 1993; Jahanshahi et al., 1992; Praamstra, Meyer, Cools, Horstink & Stegeman, 1996; Sheridan, Flowers, & Hurrell, 1987; Stelmach, Worrington, & Strand, 1986).

Individuals with PD have been noted to have difficulty carrying out movements that must be generated internally (Benecke et al., 1986; Benecke et al., 1987) and have difficulty using, or are unable to use, advance information (i.e., endogenous cues) to program movements. This has led to many investigations of the ability of individuals with PD to use informational cues to program movements. One paradigm that has been used to investigate this question contrasts a simple RT task with a choice RT task. In a simple RT task, the same response is used each time the imperative stimulus is presented, which, researchers have hypothesized, allows the individual to preprogram the response in advance of the required movement. In contrast, in the choice RT task, a different response is required depending on the actual target presented making it improbable that the response would be preprogrammed in advance of target presentation. Rather, some programming must be completed after the imperative stimulus is presented. Comparing the RT of PD subjects in an simple RT

task with that of a choice RT task is thought to provide an indication as to whether the subject was able to use the advance information to preprogram a movement. If RT in the simple RT task is faster than in the choice RT task, it is assumed that the movement in the simple RT task was preprogrammed. If RT in the simple RT task is equivalent to or slower than RT in the choice RT task, it is assumed that the subject was not able to use the information to preprogram the movement. If RT in the choice RT is slower for PD subjects than it is for control subjects, it is assumed that the PD subjects have a deficit in the speed of motor programming (Brown et al., 1993).

Some researchers have used this type of paradigm in combination with a Rosenbaum-type task where advance information is provided. Jahanshahi et al. (1992), using 15 PD and 16 control subjects (all with MMSE > 25), examined RT and MT using a simple RT and a four-choice RT task adapted from Rosenbaum (1980). All subjects were taking dopaminergic medication and were tested while on their normal dosage. The timing of their testing was not reported to be related to the time of ingestion of medication. Trials were initiated by the subject pressing down on either one or both of the home keys which elicited a central fixation cross. Visual precues were presented (an empty box or boxes) indicating hand and direction, either individually or in combinations (i.e., uncued, cued for hand only, cued for direction only, cued for

both hand and direction). These were followed at variable intervals (0ms, 200ms, 800ms, 1600ms, 3200ms) by the target, indicated by the filling in of the relevant box. Subjects were instructed to use the advance information to prepare the movement. Upon target presentation they were to lift the appropriate finger from the home key and press the target key with either the right or the left index finger as determined by the condition. PD subjects had overall slower RT than control subjects in both the simple RT and the choice RT conditions but had larger RT differences between the simple and the choice RT tasks, due to their relatively greater slowness in the choice RT task. There were no overall differences in RT between the PD and the control groups in either of the partially cued choice RT tasks. However, paired comparisons indicated that, in the fully cued task, RT was longer for the PD group than for the control group at the 200ms, 800ms and 1600ms intervals. Since RT was equivalent in the choice RT and the simple RT tasks at the 3200ms interval for both groups, the authors felt that the PD subjects were able to use advance information to plan a movement but took longer to process this information. Overall, MT was longer for PD than controls in all conditions but it was not affected differentially in any condition. This was taken to indicate that all response programming was completed prior to movement initiation. Thus, the authors suggested that PD subjects have a slowing of stimulus identification, stimulus processing,

response selection or response initiation in the choice RT situation, but if enough time is available, PD subjects are able to use cues to preprogram a movement.

Praamstra et al. (1996) also used a precuing technique in a four choice RT task. Subjects were 10 PD patients and 10 healthy controls. The PD subjects were on dopaminergic medications but testing was not reported in relation to the time of ingesting medication. Precues were presented for 1000ms and were either neutral (50%) or precued for hand (50%) (i.e., partial precue). The target stimulus was presented for 1000ms and indicated the hand and finger that were to respond. The fingers remained on response keys during the experiment (necessitating very little programming). The movement (pressing a key) was made with either the index or the middle finger. All subjects were faster to respond when provided with cues. The authors found that PD subjects had slower overall RT than controls although this did not differ proportionally between the groups (i.e., no group interaction) as a function of cue type. Thus, they concluded that, in this situation, PD subjects were able to use advance information to preprogram a movement in a similar manner as controls.

Stelmach et al. (1986) examined RT and MT using a button press task and advance precues in 8 PD and 8 control subjects. All PD subjects were on dopaminergic (and in some cases also anticholinergic) medications and the time of testing was unrelated to the medication schedule. Cues were always valid

and consisted of: 1) arm and direction, 2) arm and extent, 3) direction and extent, 4) arm, direction, and extent; 5) no cue. Cues were presented for 1000ms, followed by a 1000ms preparation interval after which the target was presented and remained illuminated until the subject responded. The subject was to press the home key to initiate a trial at which time the home key was illuminated (warning signal) and remained on during the presentation of the precues. After a one second interval, the precue lights were illuminated (for 1000ms) and were accompanied by an auditory signal. The precues were then extinguished. Following a 1000ms preparation interval, the target was illuminated. Movements were made with the index finger of either the right or the left hand. The PD subjects had overall slower RT and MT than controls but were able to use the precues to the same advantage as controls. This was indicated by their ability to respond faster with the greater amount of information provided in the precues. The authors concluded that PD patients have no difficulty in selecting the appropriate response but have a slowing of response programming and response execution compared with controls.

To examine whether the programming of movement execution and movement initiation take place at different times, Montgomery, Gorman, and Neussen (1991), using 8 PD and 8 control subjects, examined rapid wrist extension movements to changing target locations (using an apparatus where the subject's hand was placed into a wedge-shaped handle). RT and

EMG were measured. The targets consisted of LED's, positioned horizontally, to the right of the start position LED. At the auditory 'go' signal, subjects were to move their hand position to correspond with the target position illuminated. Depending on the condition, the initial target remained illuminated, or, at various intervals, was extinguished and a secondary target was illuminated. This gave the appearance of the initial target as having moved. Seven conditions were used where the target was either moved or not moved. Conditions were, target moved: 1) 500ms prior to the go signal, 2) simultaneously with the go signal, 3) 200ms after the go signal, 4) at movement onset, 5) 100ms after movement onset, 6) 200ms after movement onset, or 7) target not moved. Feedback regarding hand position was provided by LED's which displayed current hand position relative to the targets. RT was determined by the handle moving. PD subjects were tested twice. On one testing session they had fasted overnight from ingesting their regular medication and on one testing session they were tested one hour after having ingested their regular medications (i.e., once on and once off medications). The types and dosages of medications differed according to the individual but included L-dopa and carbidopa for all subjects. PD subjects had longer RT compared with controls, possibly due, according to the authors, to reduced efficiency in motor recruitment. Results revealed that both groups made single trajectories if target moved prior to movement initiation and

double trajectories if the target did not move or moved at or after movement onset. Single trajectories consisted of smooth wrist movements with a single agonist EMG burst followed by a maintained level of EMG activity during extension. Double trajectories consisted of 2 separate movements, the first movement reaching the initial target and the second movement reaching the secondary target. In double trajectories, the agonist EMG consisted of a double peak followed by a maintained level. In both types of trajectories, the initial EMG agonist burst was followed by an antagonist burst which was initially high but reduced prior to movement initiation. The authors interpreted this to indicate that the trajectory had been programmed prior to the initiation of the movement by all subjects.

The studies reviewed above have concluded that although individuals with PD are slower to initiate and execute responses than control subjects, they are able to use advance information (precues) from the environment to plan movements as well as control subjects. However, a number of studies do not support this claim, instead suggesting that individuals with PD are either unable to use, or have difficulty using, advance information to preprogram movements.

Bloxham, Mindle, and Firth (1984) examined RT in a task where subjects were to respond to the commands "go left" or "go right" by lifting the corresponding index finger from a button (experiment 2). Precues consisted of the words "ready

left" (valid), "ready right" (valid), or "ready" (neutral). While both groups were able to benefit from the valid cues, the PD group (n = 9, all on dopaminergic medication) benefited significantly less than the control group (n = 11) even though they performed equivalently to the controls in the neutral condition. Bloxham et al. (1984) concluded that PD patients have difficulty using advance information to select or initiate the appropriate movement.

Sheridan et al. (1987) examined simple RT and choice RT tasks using aiming movements (moving a lever with the hand to meet a target on the screen). A "no aim" condition was also used where the subject was instructed to move as quickly as possible to the stimulus but not to aim at it. The authors found that PD subjects were able to initiate a movement as quickly as controls (choice RT) from a start position but were not able to use advance information (simple RT) to reduce RT. Both groups had equivalent RT and MT in the neutral ("no aim") condition although the controls had higher velocity. Overall MT was approximately 37% slower for PD subjects than controls due to a significantly longer MT for the first half (i.e., total distance of movement divided in half) of the movement than controls.

In summary, as is evident from the above reviewed literature, there is no clear consensus regarding whether individuals with PD are able to use precues to preprogram a movement in a manner similar to healthy controls but most

evidence suggests that they are.

Covert Orienting of Attention in PD

The interest in covert orienting of attention in PD follows from results of animal studies which have revealed a strong association between the availability of dopamine in the striatum and visual orienting of attention (Clark, Geffen & Geffen, 1987). Since PD is known to reduce levels of dopamine in the striatum, this disorder appears to be an appropriate naturalistic model to study the effect of dopamine depletion on visual orienting of attention in humans. The investigation of covert shifts of visual attention in PD has been examined using RT paradigms almost exclusively.

Wright, Burns, Geffen, and Geffen (1990) examined covert orientation of attention in PD patients using a Posner-type RT paradigm in which an endogenous cue (valid, invalid, neutral) was followed (after 1100ms) by an exogenous target. Results revealed that PD subjects had overall slower RT than controls, had equivalent "benefits", and had reduced "costs" compared to controls. This was interpreted, by the authors, to indicate that PD subjects had no deficits in the covert movement and engagement of attention but had a deficit in the ability to maintain attention. Similar results were found by Wright, Cremona-Meteyard, Geffen, and Geffen (1994). They examined covert orienting of attention by PD patients using a RT paradigm in which subjects were required to push a button in response to the presentation of a peripheral target presented

to either the right or left of centre. The target was preceded (by 100ms) by an endogenous cue that was either valid, invalid, or neutral. Results revealed that PD subjects had similar "benefits" but reduced "costs" compared with controls suggesting to the authors that there is a deficit in PD patients' ability to maintain attention (i.e., they fail to maintain their attention on an invalidly cued location).

Bradshaw, Waterfall, Phillips, Iansek, Mattingley, and Bradshaw (1993), (experiment 3) examined covert orienting using a vibrotactile paradigm combined with a Posner-type paradigm. Twelve PD subjects (all on antiparkinsonian medication) and 12 control subjects were included. The target stimulus was a tactile stimulation to either the left or the right index finger. They were instructed to press a button as quickly as possible with the stimulated finger when the stimulus was presented. Cues were valid, neutral and invalid and consisted of a 200ms burst of rapidly alternating and cycling frequencies that was different from the stimulus. The SOA was 300ms. Neutral trials were administered in separate blocks from the informational cues. Overall RT was slower for the PD than the control group. Post-hoc paired comparisons of the three cue types indicated that, for the PD group, none of the cues differed from one another. In contrast, the control group had both significant "costs" and "benefits". Although there were no significant differences among the cue types for the PD group, the authors examined the

magnitude of the differences between the valid and neutral conditions and the invalid and neutral conditions for the two groups. They concluded that since the benefit was 45ms for the PD group and 52ms for controls and the costs were 24ms for the PD group vs 44ms for the controls, that the PD group had reduced costs and therefore a difficulty in the maintenance of attention.

In contrast to the findings of Bradshaw et al. (1993) and Wright et al. (1990, 1994), other researchers have noted no deficit in the maintenance of attention for individuals with PD. For example, Rafal, Posner, Walker, and Friedrich (1984) examined the covert orientation of attention (experiment 2) in nine PD subjects. There were no control subjects. Rather, the PD subjects served as their own controls and were tested when "on" and "off" their drug therapy (L-dopa and carbidopa). An exogenous cue was used where the subject fixated on a central cross, and one of the two peripheral boxes to the left or the right of fixation brightened. The cue was valid 80% of the time and invalid 20% of the time. No neutral cues were used. The target appeared at variable intervals after the cue (50ms, 150ms, 500ms, 5000ms). There was a general decline of RT with increasing SOA. RT when "on" medication was generally faster than when "off" medication. There was no interaction of SOA or medication status with cue validity. The group means of the subject's median RT's indicated that PD subjects were able to benefit from valid cues compared with invalid cues (i.e., RT

faster for valid cues) at all SOA's indicating, according to the authors, that PD subjects had no difficulty shifting attention, and presumably no difficulty maintaining it either.

Bennett, Waterman, Scarpa, and Castiello (1995), using a Posner-type paradigm, investigated covert shifts of attention in 32 PD patients and 32 control subjects. Endogenous cues (neutral, valid, invalid) were used. Trials began with a central fixation cross appearing on the screen with two empty boxes (one left; one right). After 500ms, a cue appeared above the central fixation cross, followed 600ms later by a red dot that appeared in the target box. Subjects were instructed to press the space bar as quickly as possible upon target presentation. PD subjects had overall longer RT's than controls but had equivalent "costs" and "benefits" to controls suggesting that the PD subjects did not have deficits in either orienting or maintaining attention.

Sharpe (1990) investigated the covert orienting of attention in 20 PD patients and 20 controls. Endogenous cues were used (neutral, valid, or invalid). Subjects were instructed to maintain their gaze on a central fixation point and to press a button with either the left or the right index finger upon target presentation. Results indicated that PD subjects had slower overall RT than controls. Both groups showed equivalent "benefits" and no "costs". The authors suggest that the increased RT for PD subjects was due to the decision process of the movement rather than to the motor

component of the task because there was no significant correlation between the clinical signs score (a neurological scale with rating of 0-4) and RT in each of the cue conditions for the PD subjects.

Yamada, Izyuuinn, Schulzer, and Hirayama (1990) examined the covert orientation of attention using an RT task and two groups of PD patients (all on anti-Parkinsonian medications). Group 1 had a Hoehn and Yahr score of I or II and group 2 had a score of III or IV. All were reportedly within the normal range on the MMSE (although no scores were provided). Subjects in group 2 were significantly older (mean = 68.7 years) than those in group 1 (mean = 58.5 years) and had PD significantly longer (mean = 10.1 years) than group 1 (mean = 4.3 years). In each trial, a central directional arrow was presented that indicated which field (left, right) to attend to. Each field contained 3 possible target locations. Simultaneously with the central directional arrow, one of the three boxes in the cued hemifield brightened (the cue). After a 100ms interval, a red cross appeared in the target box and the subject was instructed to press a button as quickly as possible with the right index finger upon target presentation. There were three types of invalid cues in each visual field and a crossed invalid condition where the target appeared in the opposite hemifield (in the box that was in the same position as was cued in the other hemifield). Results indicated that the PD subjects in group 2 had the longest overall RT. The control

subjects and the PD subjects in group 1 showed significant RT differences between the valid and the crossed invalid conditions (valid < invalid) while these were equivalent for the group 2 PD subjects. This paradigm differed from others that have investigated covert orienting (e.g., Bennett et al., 1995; Rafal et al., 1984; Wright et al. 1990, 1994) in that it paired simultaneous endogenous and exogenous cues. The authors interpreted the results to indicate that the ability to shift attention is very weak or nonexistent for more severely impaired PD patients but is intact in the earlier stages of PD.

In summary, research on the covert orienting of attention in PD has noted there to be no deficit in the shift or engagement of attention (Rafal et al., 1994; Wright et al., 1990), at least until the later stages of the disease (Yamada et al., 1990). It has been suggested that there is a deficit in the maintenance of attention in PD in which they disengage more quickly from a target compared with controls (Bradshaw et al., 1993; Wright et al., 1990, 1994) although not all studies support this (Bennett et al., 1995; Sharpe, 1990). All of the studies examining covert orienting of attention in PD have referred to reduced "costs" as a "deficit" in the ability to maintain attention (in comparison to healthy elderly subjects). However, this "deficit" might also be thought of as a benefit that allows the PD subject to disengage from the invalid target more quickly and, thus, complete the action

more quickly.

Some researchers have suggested that PD subjects rely more on external cues (analogous to exogenous overt orienting in the current study) than on internal cues (analogous to endogenous overt orienting in the current study) to control movements. Numerous types of paradigms have been used to examine this latter issue.

Internal vs External Cuing in PD

Bradshaw, Merran, Waterfall, Phillips, Iansek, Mattingley, and Bradshaw (1993) conducted three experiments investigating covert orienting and endogenous (i.e., internal) versus exogenous (i.e., external) cuing using a vibrotactile paradigm with 12 PD (all on antiparkinsonian medication) and 12 control subjects. In the first experiment, subjects maintained their gaze at a central fixation point, and received a tactile stimulation to either the left or the right index finger. They were instructed to press a button as quickly as possible with the stimulated finger when the stimulus was presented. They were told in advance of the block of 12 trials on which hand to expect the stimulus (75% expected; 25% unexpected). The authors considered this type of cuing to be internal cuing (i.e., subjects had to keep it in mind throughout the block of trials). Results revealed that the control subjects had faster RT to the expected than to the unexpected hand while PD subjects showed no difference between the two conditions (due to slower responses to expected trials

than the control subjects). The authors interpreted this to indicate that the PD subjects had difficulty maintaining attention to expected stimuli. In the second experiment, the task was identical to the first with the exception that the subjects were instructed to either look at or look away from the expected hand (an overt shift of gaze which was associated with a redirection of attention). They were to keep their gaze on the tip of either the right or the left index finger. Once again, PD subjects had no effect of stimulus expectancy due to slower responses in comparison to controls to the expected hand. However, PD subjects did have faster RT when looking at the expected hand than when looking at the unexpected hand, leading the authors to suggest PD subjects may rely more on overt (external) than on covert (internal) shifts of attention (which, in this study were accomplished by a shift of gaze manipulation). They suggest this is due to PD subjects being less able to "maintain a mental set against competing alternatives" (p. 56) but that external cues help them to do so.

Kritikos, Leahy, Bradshaw, Iansek, Phillips, and Bradshaw (1995) also examined "internal" and "external" control of movement. Subjects were 12 PD patients (all but one were taking antiparkinsonian medication) and 12 healthy controls. They used a task where subjects were required to press a series of buttons in sequence as quickly and accurately as possible. Two conditions were used: with visual cues (the

target lights remained illuminated; external cues), and without visual cues (the target lights were not illuminated and the subject had to remember the movement sequence; internal cues). Results indicated that PD subjects had overall slower RT than controls. As well, PD subjects moved from button to button more slowly when the visual cues were removed versus when they were present. In contrast, control subjects showed no differences between the visual and no visual information conditions. The authors concluded that PD subjects perform better when external cues are available (i.e., overt shifts of attention) than when having to rely on internal cues (i.e., covert shifts of attention).

Brown, Schwarz, Bowman, Fuhr, Robinson, and Hallett (1993) examined the use of internal versus external information in PD using 3 variations of a choice RT task. The task was to lift the left or right index finger when the target was indicated. Targets were: 1) spatially compatible (a box presented on the left or right of fixation); 2) compatible symbolic (a centrally presented arrow pointing to either the left or the right); and 3) arbitrary symbolic (circle indicated left response; square indicated right response). Overall RT was slower for PD subjects than controls but they were not differentially impaired when using internal versus external cues.

In summary, the literature reviewed in this chapter has revealed some consistencies but also somewhat conflicting

results. Individuals with PD have been noted to have generally slower RT and longer MT compared with controls (Brown et al., 1993; Jahanshahi et al., 1992, 1993; Muller & Stelmach, 1992; Praamstra et al., 1996) and have also been able to use cues to facilitate performance (Bloxham et al., 1984; Praamstra et al., 1996; Wright et al., 1990, 1993, 1994). However, they may take longer to use the cued information than controls (Jahanshahi et al., 1992) or have more difficulty using the cued information compared with controls (Bloxham et al., 1994). Some researchers have also suggested that individuals with PD are not able to use such cues to facilitate performance (Sheridan et al., 1987).

It has generally been considered that persons with PD do not have a deficit in the ability to shift attention covertly (Rafal et al., 1994; Sharpe, 1990; Wright et al., 1990, 1993, 1994), at least not until later stages of the disease (Yamada et al., 1990). Rather, individuals with PD have been noted, by some, to have a deficit in the ability to maintain attention (Bradshaw et al., 1993; Wright et al., 1990, 1993, 1994; Yamada et al., 1990), although this has not been a consistent finding (Bennett et al., 1995). It has been suggested that individuals with PD may rely more on, or be better able to use, external information compared with internal information (Bradshaw et al., 1993; Brown & Marsden, 1988; Kritikos et al., 1995) although there have been exceptions to this also (Brown et al., 1993).

Since these conclusions often come from separate approaches to the study of PD (kinematics, motor programming, orienting of attention) and have subsequently used numerous types of paradigms, the lack of a clear consensus based on the results is perhaps not surprising. There appear to have been no studies to date that have attempted to combine the orienting of attention, use internal and external cues, and use kinematic analysis of motor programming in examining visuomotor integration and motor execution in individuals with PD.

Parkinson's Disease vs Alzheimer's Disease

There is one study that has examined the orienting of attention in PD compared with AD. This was conducted by Caffarra, Riggio, Malvezzi, Scagloni, and Freedman (1997). Subjects included AD (n = 7), PD without dementia (n = 10), PD with dementia (n = 7) and normal controls (n = 10). A Posner-type paradigm with central cues and valid and invalid trials (to the left or right of centre) was used. Subjects were to press a key as quickly as possible when the target was detected. Results indicated that all groups responded faster to valid than invalid cues and there was no group by cue validity interaction evident in the four-way analysis of variance (using a logarithmic transformation of the data). Overall, RT was faster for the PD without dementia group than the PD with dementia group, and all groups had slower RT than control subjects. No other significant RT differences were

discussed, and, thus, presumably none were found. The data they presented for RT indicated that the mean value (of the median RT's) for the PD group was faster than for the AD group. However, the variability was large for both groups and may have been responsible for no significant differences being found.

Another study, while including both AD and PD subjects, did not compare the two groups statistically. Wright, Cremona-Meteyard, Geffen, and Geffen (1994) included subjects with AD (n = 11), PD (n = 20), and two groups with closed head injuries (mild, n = 9; moderate to severe, n = 11) in their study. Centrally presented valid, invalid, and neutral cues were used. Subjects were to press a key when the target was presented. Each group had its own control group, and statistical comparisons were confined to each clinical group with its control group. Extracting mean RT values from the data they presented for the various groups indicated an overall faster mean RT for PD than AD subjects but it is not possible to determine whether this difference is significant.

Cossa, Della Salla, and Spinnler (1989) used AD (n = 24) subjects, PD (n = 25) subjects, and elderly controls (n = 30) in a paradigm where the subject was to detect the letter "T" from a circular display of letters. The "T" was either presented in 1) a random position, 2) a selected position or, 3) in a primed position. The required response was a button press. RT and accuracy were examined. No differences in

accuracy were found among the three groups. The PD group had faster RT than the AD group but slower RT than the control group.

These three studies do not provide conclusive evidence regarding the differences in RT between AD and PD subjects. There is evidence that the values of RT may be faster for PD than AD subjects but whether these values differ significantly is questionable. One study suggests that accuracy may be equivalent for the AD and PD groups.

HYPOTHESES

Cue Effects for Parkinson's Subjects

PD subjects were expected to show faster RT, shorter MT, and higher PV to valid than invalid cues for both paradigms. They were expected to have RT "costs plus benefits" for both paradigms and MT "costs plus benefits" for the Endogenous Paradigm. PD subjects were expected to show an advantage of the Exogenous over the Endogenous Paradigm.

Parkinson's Disease Subjects vs Elderly Control Subjects:

PD subjects were expected to have slower RT, longer MT, and lower PV than elderly control subjects. PD subjects were expected to have smaller "costs plus benefits" than the elderly control group for the Exogenous Paradigm (based on the literature that suggests they have a deficit in the maintenance of attention that results in lower "costs"). PD subjects were expected to have greater "costs plus benefits" than the elderly control subjects for the Endogenous Paradigm

due to reported difficulties with motor programming.

Parkinson's Disease Subjects vs Alzheimer's Disease Subjects:

It was difficult to predict, from the literature, whether RT for the PD group would differ from that of the AD group. This was due to few studies making such comparisons and the fact that the studies that have examined this have used a button press as a response. Given that the response in this thesis was an aiming movement to a target, it seemed likely that PD subjects (who are known to have a motor disorder) would have longer RT than those with AD. PD subjects were expected to have longer MT and lower PV than AD subjects. PD subjects were expected to have smaller RT "costs plus benefits" for the Exogenous Paradigm than the AD group.

CURRENT STUDY

This study attempted to address the differences in the literature discussed above and compared the performance of subjects with PD to subjects with AD and elderly control subjects. The current study used the same two experimental paradigms described previously for the young, healthy elderly, and AD subjects.

METHOD

Subjects

Subjects were 11 individuals (6 males; 5 females) who had a diagnosis of idiopathic PD. The gender breakdown did not differ significantly between the AD and the PD groups (chi-square = 0.05, p = .82). Subjects were recruited from the

Fetal Transplantation Program of the Queen Elizabeth II Health Sciences Centre (none had undergone surgery at the time of their participation in this study) and from the practice of Dr. D. King, neurologist. All diagnoses had been made by neurologists. Mean age was 68.9 (SD = 9.69), with a range of 49 to 79 years. Hoehn and Yahr scores ranged from 2.0 to 3.0 with a mean of 2.5 for the group and were determined by the patients' neurologist. Mean level of education was 13.5 years (SD = 3.2) with a range of 7 to 18 years. Mean MMSE score for the PD group was 26.8 (SD = 1.9) with a range of 23 to 29. None of the PD subjects were considered to meet DSM-IV criteria for dementia by their clinicians. The mean MMSE score was higher for the PD group than the AD group (chi-square = 8.70, $p < .005$) and lower than that of the elderly control group (chi-square = 21.3, $p < .0001$). Mean age and education for the PD group did not differ significantly from the elderly or the AD groups (all chi-square p values > .05). Table 7.1 illustrates the demographics of the PD group including the MMSE and Hoehn and Yahr scores. Informed consent was obtained from all subjects prior to testing in accordance with the procedures approved by the Queen Elizabeth II Health Sciences Centre Research Ethics Committee.

Table 7.1

Parkinson Subject Demographics

Subject	Age	Sex	Educ	MMSE	Hoehn-Yahr
1	49	M	13	29	2.0
2	76	M	7	23	2.0
3	66	M	14	27	2.0
4	76	F	14	27	2.0
5	71	M	18	26	2.5
6	62	F	14	28	2.5
7	79	F	11	24	2.5
8	71	F	12	27	2.5
9	56	M	16	29	3.0
10	74	F	11	27	3.0
11	78	M	18	28	3.0

Procedure

The procedure for the experiments was identical to that of the other three groups. All PD subjects were tested within two hours after ingesting their regular medication in order that they would be tested during a period in which their signs and symptoms of Parkinson's Disease would be least. Specific details on medication type and dosages were not collected. However, all subjects were taking optimal antiparkinsonian medication as judged by their neurologists.

ANALYSES

Analysis 1 was a 3 (Cue Condition - Valid, Invalid, Neutral) X 2 (Target Type - Exogenous, Endogenous) repeated measures MANOVA using SAS General Linear Model procedures. This analysis was for Parkinson's disease subjects only. The dependent measures were reaction time (RT), movement duration time (MT), peak velocity (PV), percent deceleration and resultant error.

Analysis 2 was a 3 (Cue Condition - Valid, Invalid, Neutral) X 2 (Target Type - Exogenous, Endogenous) X 3 (Group - Elderly Control, AD, PD) repeated measures MANOVA using SAS General Linear Models procedures. The dependent measures were reaction time (RT), movement duration time (MT), peak velocity (PV), and percent deceleration. Resultant error was not included in the MANOVA as the MANOVA will only use those trials for which there is data for all variables. Due to the tremors exhibited by some of the subjects in the PD group, it

was difficult to establish end of motion by the computer algorithm (velocity < 5cm/sec). Although most often it was possible to establish end of motion on these trials by a visual inspection of the raw velocity data files, resultant error could not be obtained from these raw data files. Since missing data on any one variable on a particular trial results in all data from that trial being excluded in the MANOVA program, resultant error was not included in the MANOVA in order to increase power for comparisons on other dependent measures. Instead, a separate 3 X 2 X 3 ANOVA was conducted on the resultant error dependent variable for the three groups.

A series of a priori planned paired comparisons using the least squares adjusted means was conducted to examine the differences in the "costs plus benefits" (i.e. Invalid minus Valid) for each of the dependent variables.

ANALYSIS I

Parkinson's Disease Subjects

The MANOVA revealed a multivariate main effect of cue condition, $F(8, 36) = 4.52$, $p < .001$, and of target type $F(4, 7) = 13.17$, $p < .005$. There was no multivariate cue condition by target type interaction, $F(8, 36) = 1.25$, $p = .30$.

Reaction Time (See Figure 7.1)

Results of the univariate mixed model ANOVA revealed a main effect of cue condition for RT, $F(2, 20) = 11.29$, $p < .0005$. Examining the least squares adjusted means revealed that the valid condition (M = 688.2ms, SE = 16.0) was

marginally faster than the invalid condition (*M* = 744.6ms, *SE* = 27.4ms; *t* = 1.78, *p* = .09) and the neutral condition (*M* = 838.1ms, *SE* = 27.5ms; *t* = 4.71, *p* < .0001). The invalid and neutral conditions differed only marginally (*t* = 2.41, *p* = .05).

There was a univariate main effect of target type, *F* (1,10) = 5.87, *p* < .05. The Exogenous Target Paradigm had a significantly faster average RT (*M* = 652.5ms, *SE* = 60.8ms) than the Endogenous Target Paradigm (*M* = 861.3ms, *SE* = 61.1ms; *t* = 2.42, *p* < .05). There was no univariate cue condition by target type interaction, *F*(2, 20) = 2.15, *p* = .14. "Costs plus benefits" for the Exogenous Paradigm was significant (88.1ms. *t* = 3.01, *p* < .01) while the "costs plus benefits" for the Endogenous Paradigm were not (24.6ms, *t* = 0.83, *p* = .41).

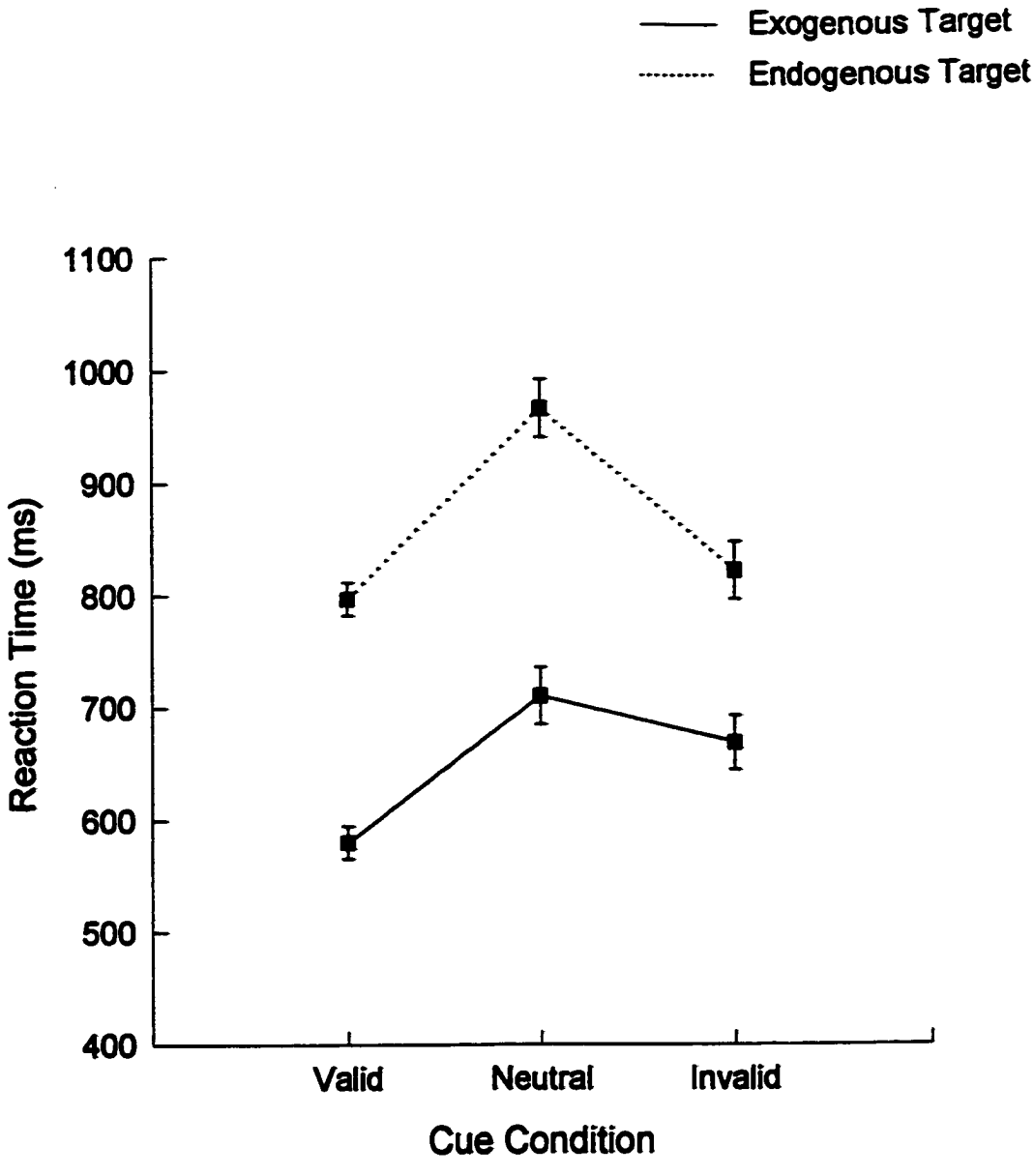


Figure 7.1 - Reaction time as a function of cue condition and target paradigm. The error bars represent the standard error of the least squares means.

Movement Duration (See Figure 7.2)

Results of the univariate mixed model ANOVA revealed a main effect of cue condition, $F(2, 20) = 9.76$, $p < .001$. Examining the least squares adjusted means revealed that the valid condition (M = 766.7ms, SE = 5.6) was significantly faster than the invalid condition (M = 804.5ms, SE = 9.5ms; $t = 3.42$, $p < .005$) and the neutral condition (M = 806.1ms, SE = 9.6ms; $t = 3.56$, $p < .002$). The invalid and neutral conditions were equivalent ($t = 0.11$, $p = .91$).

There was no main effect of target type, F (1,10) = 2.78, $p = .13$ for MT. There was also no univariate cue condition by target type interaction, $F(2, 20) = 1.30$, $p = .30$. A priori planned comparisons revealed significant "costs plus benefits" for both the Exogenous Target Paradigm (27.8ms, $t = 2.85$, $p < .01$) and for the Endogenous Target Paradigm (48.0ms, $t = 4.91$, $p < .0001$).

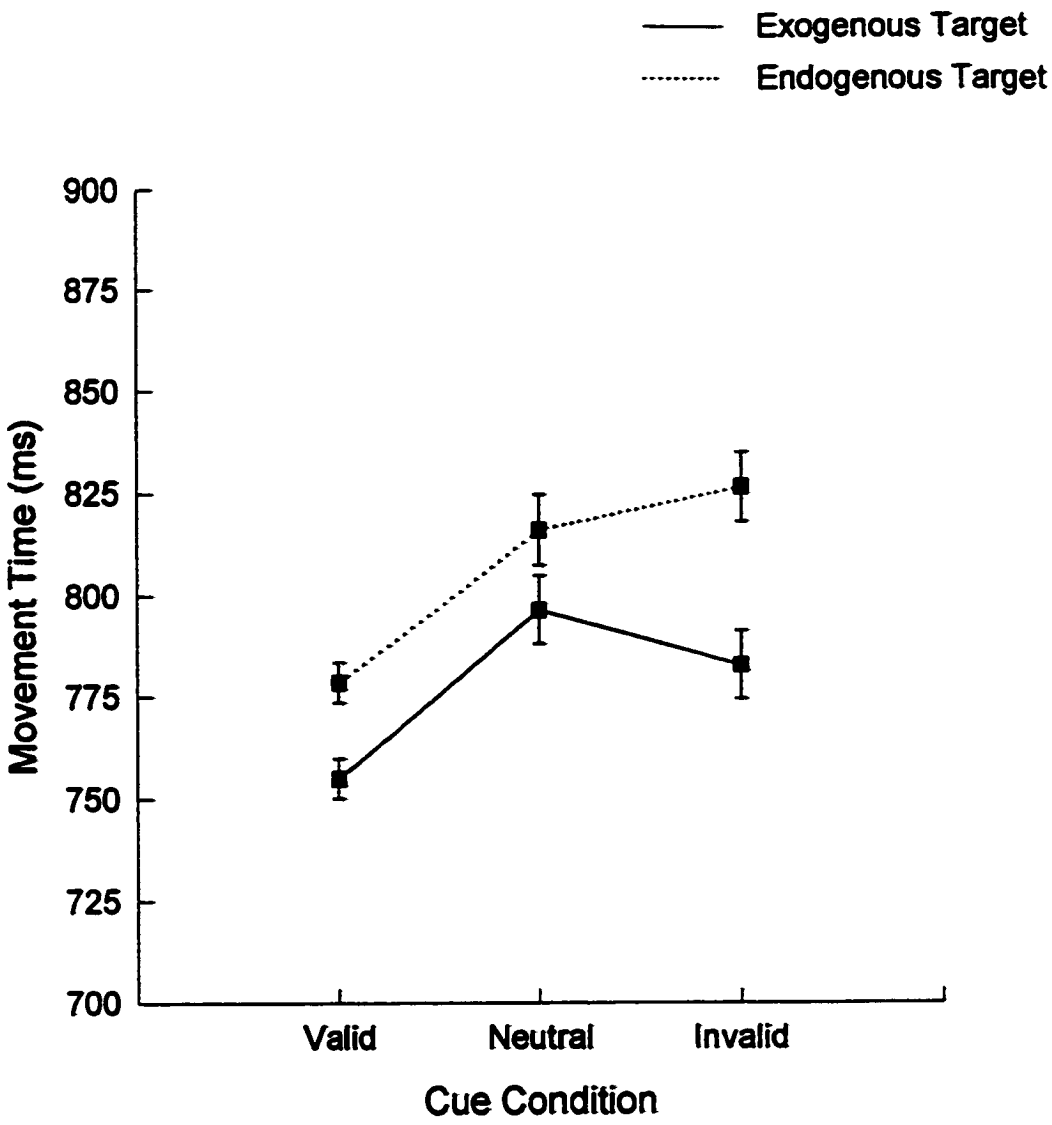


Figure 7.2 - Movement time as a function of cue condition and target paradigm. The error bars represent the standard error of the least squares means.

Peak Velocity (See Figure 7.3)

Results of the univariate mixed model ANOVA revealed no main effect of cue condition for PV, $F(2, 21) = 1.21$, $p = .32$. There was a univariate main effect of target type, $F(1,10) = 21.7$, $p < .001$. The Exogenous Target Paradigm had a significantly higher PV (M = 65.9cm/sec, SE = 0.54cm/sec) than the Endogenous Target Paradigm (M = 62.3cm/sec, SE = 0.54cm/sec, $t = 4.65$, $p < .005$). There was no univariate cue condition by target type interaction, $F(2, 21) = 2.44$, $p = .11$. There were no significant "costs plus benefits" for either of the two target paradigms.

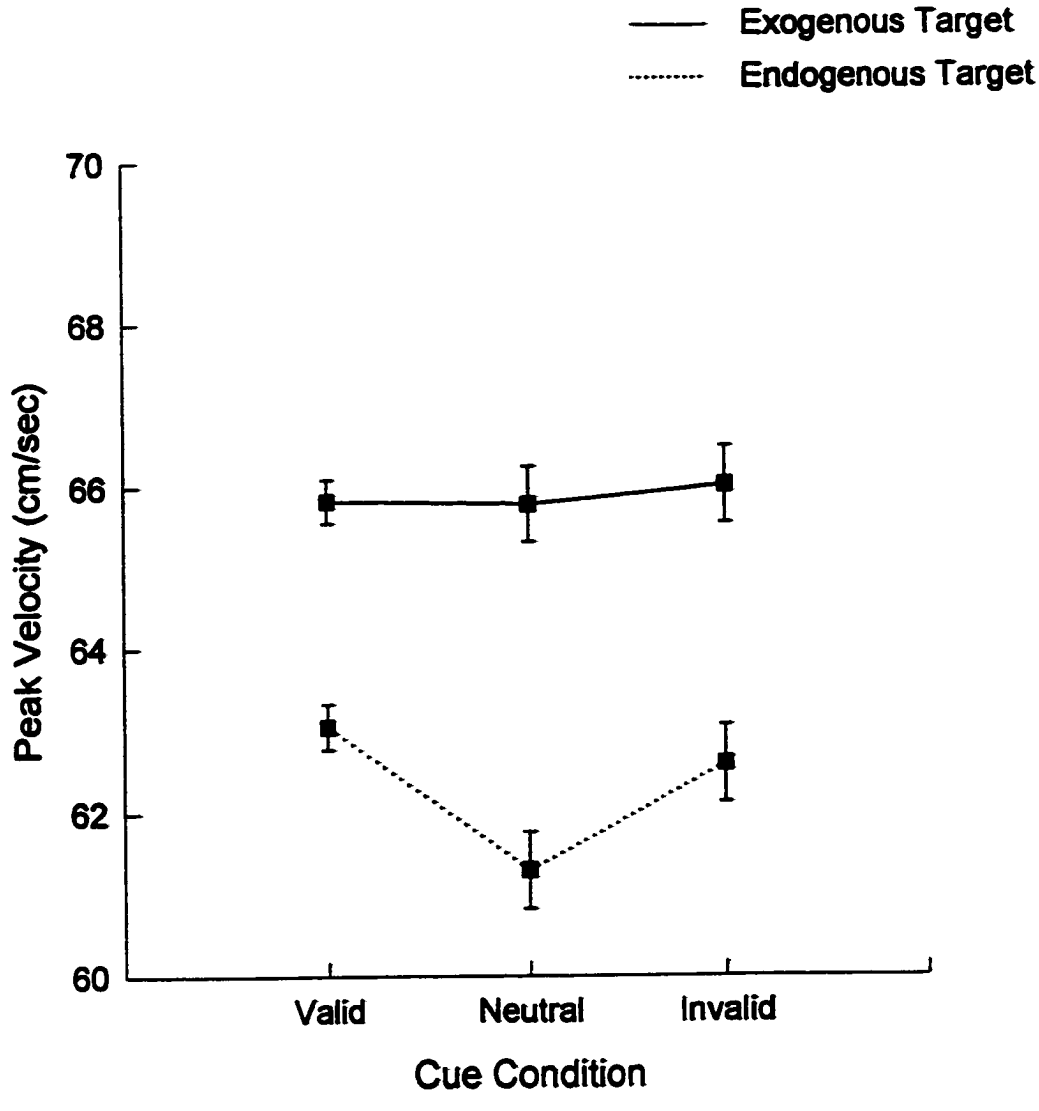


Figure 7.3 - Peak velocity as a function of cue condition and target paradigm. The error bars represent the standard error of the least squares means.

Percent Deceleration (See Figure 7.4)

Results of the univariate mixed model ANOVA revealed no main effect of cue condition for percent deceleration, $F(2, 20) = 0.49$, $p = .62$. There was a marginally significant univariate main effect of target type, $F(1,10) = 4.38$, $p = .06$. The Exogenous Target Paradigm had a marginally greater percent deceleration ($M = 59.6\%$, $SE = 0.53\%$) than the Endogenous Target Paradigm ($M = 57.9\%$, $SE = 0.53\%$, $t = 2.12$, $p = .06$). There was no univariate cue condition by target type interaction, $F(2, 20) = 0.42$, $p = .66$. There were no significant "costs plus benefits" for either target paradigm.

Resultant Error (See Figure 7.5)

Results of the univariate mixed model ANOVA revealed no main effect of cue condition for resultant error, $F(2, 21) = 1.32$, $p = .29$ or of target type, $F(1,10) = 0.27$, $p = .61$. There was no univariate cue condition by target type interaction, $F(2, 21) = 1.47$, $p = .25$. There were no significant "costs plus benefits" for either target paradigm.

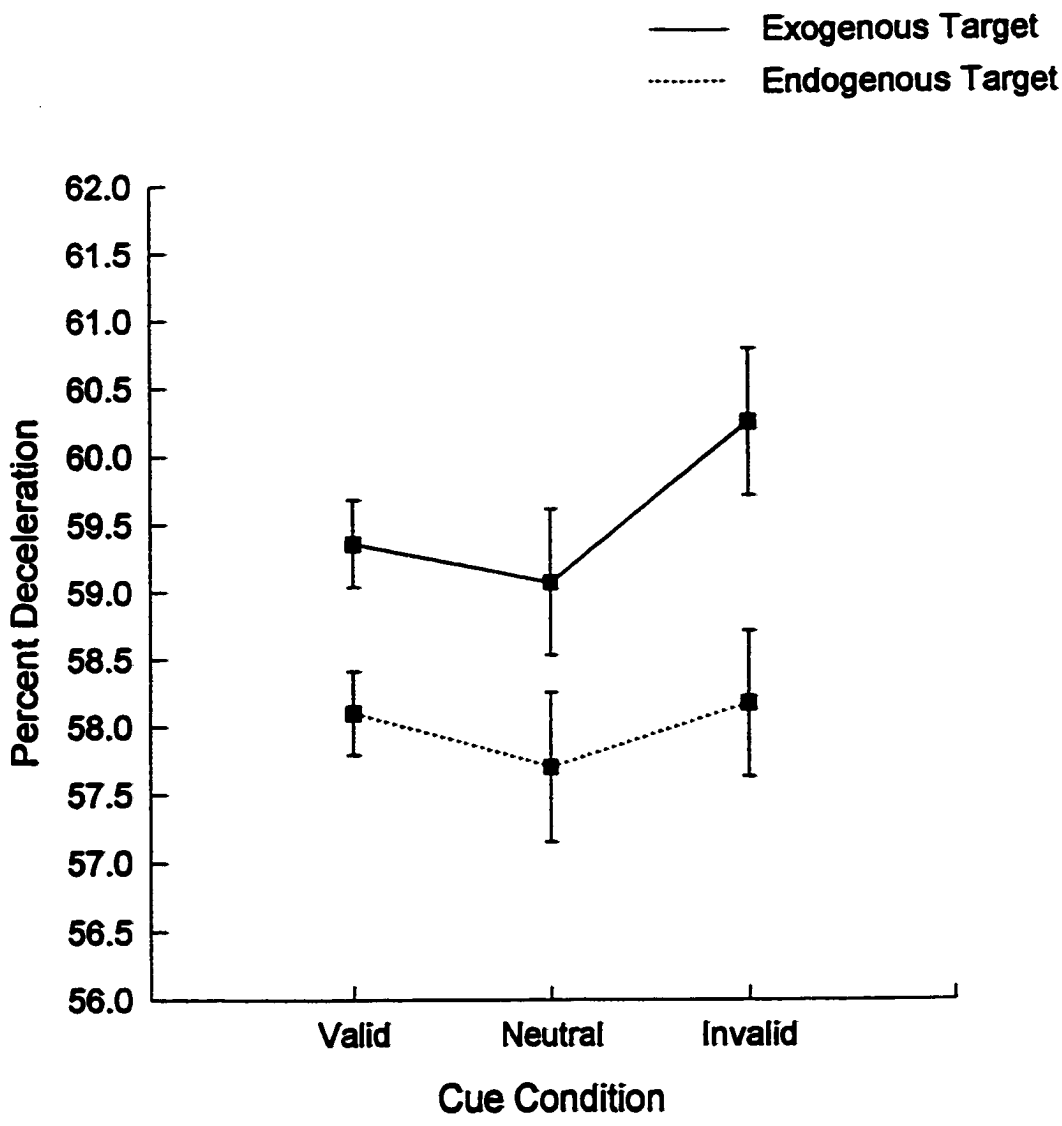


Figure 7.4 - Percent deceleration as a function of cue condition and target paradigm. The error bars represent the standard error of the least squares means.

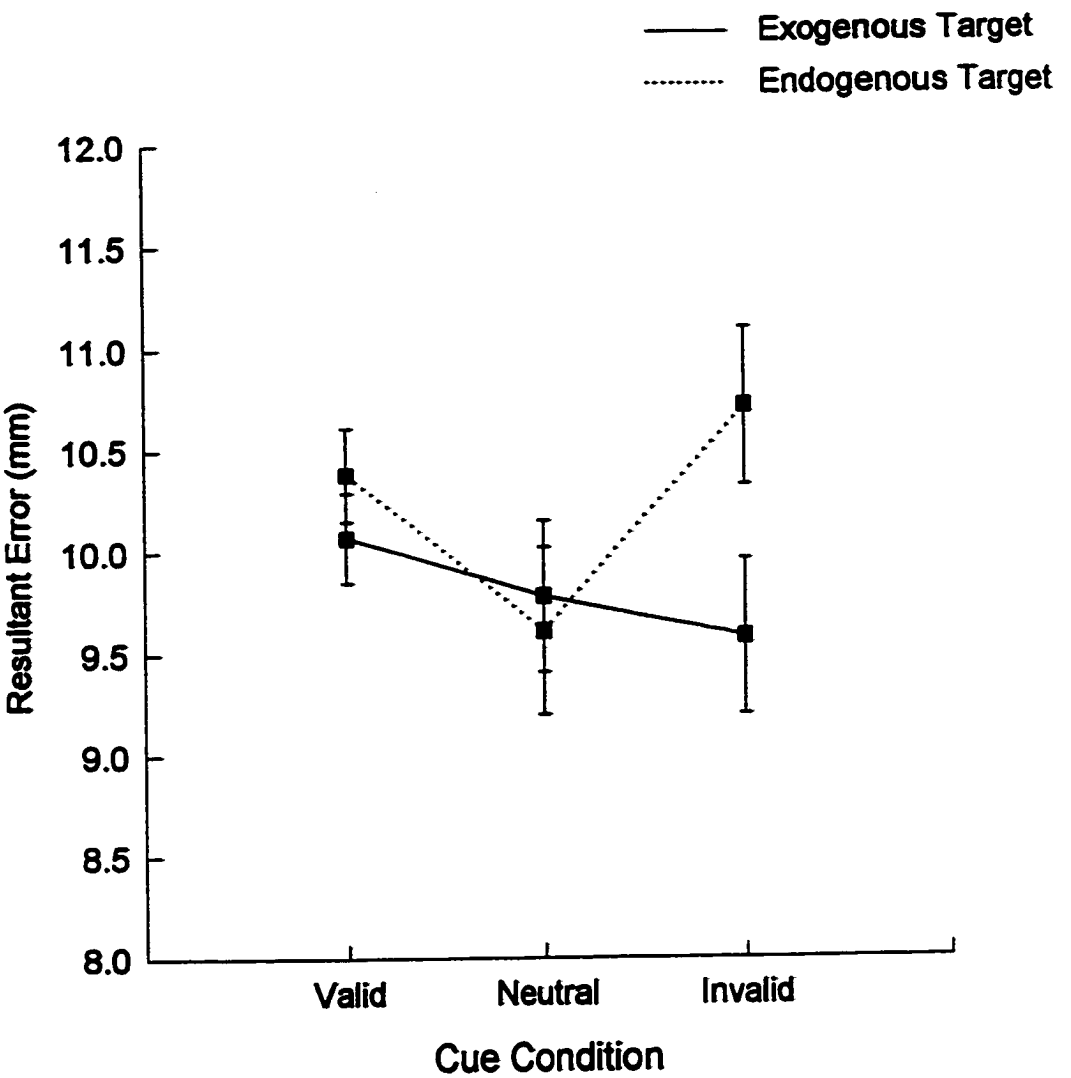


Figure 7.5 - Resultant error as a function of cue condition and target paradigm. The error bars represent the standard error of the least squares means.

ANALYSIS 2

Elderly Control, Alzheimer's and Parkinson's Disease Groups

The MANOVA revealed a marginal multivariate main effect of group, $F(8, 62) = 1.89$, $p = .07$, and multivariate main effects of cue condition $F(8, 128) = 11.77$, $p < .0001$, and of target type $F(4, 30) = 11.77$, $p < .0001$. In addition, there was a multivariate group by condition interaction, $F(16, 264) = 2.52$, $p < .001$, and a multivariate condition by target type interaction, $F(8, 128) = 2.63$, $p < .01$. There was no multivariate group by target type interaction, $F(8, 62) = 1.71$, $p = .11$, and no multivariate group by condition by target type interaction, $F(16, 264) = 1.41$, $p = .13$. The MANOVA was followed up with a univariate mixed model Analysis of Variance (ANOVA) for each of the dependent variables.

Reaction Time (See Figure 7.6)

Results of the univariate mixed model ANOVA revealed a main effect of group for RT, $F(2, 33) = 4.32$, $p < .02$. The AD and PD groups differed marginally ($t = 1.72$, $p = .09$), with the PD group having an average RT which was 226ms faster than the AD group (collapsed across cue condition and target type). The PD and the elderly control groups did not differ significantly from one other ($t = 1.13$, $p = .26$) although the pattern was for the elderly subject group to have a faster RT than the PD group (624.8ms vs 756.9ms, respectively). The AD and elderly controls differed significantly ($t = 2.91$, $p < .01$) with the elderly control group having an average RT that

was 357ms faster than the AD group (624.8ms vs 981.8ms, respectively).

There was a group by cue condition interaction, $F(4, 66) = 5.94$, $p < .0005$, due to different patterns of performance among the three groups across cue conditions. Paired comparisons of the least squares adjusted means indicated that, for the PD group, RT in the neutral condition (M = 838.1 ms, SE = 28.7ms) was significantly slower than that for the invalid condition (M = 744.6, SE = 28.5; t = 2.31, $p < .02$) and the valid condition (M = 688.2ms, SE = 16.7ms; (t = 4.51, $p < .0001$). There was only a marginally significant difference between the invalid and the valid conditions (t = 1.70, p = .09) with the valid condition being faster than the invalid condition. For the AD group, the invalid and neutral conditions did not differ significantly (t = 1.26, p = .23) while the valid condition was faster than the invalid condition (t = 6.30, $p < .0001$) and the neutral condition (t = 7.72, $p < .0001$). For the elderly subject group, the invalid and neutral conditions did not differ (t = 0.65, p = .52) while the valid condition was significantly faster than the invalid (t = 2.91, $p < .005$) and neutral conditions (t = 3.71, $p < .0005$). Thus, the pattern for the AD and the elderly group was similar (invalid = neutral; valid faster than invalid and neutral). The only difference for the PD subjects was that they had a faster RT for the invalid than the neutral condition. There were also magnitude differences among the

groups with the AD group having greater differences between the conditions than the PD and elderly control groups.

There was a main effect of target paradigm, $F(1, 33) = 28.8$, $p < .0001$. The Exogenous Paradigm had a RT that was an average of 206.7ms faster than the Endogenous Paradigm ($t = 5.34$, $p < .0001$) (collapsed across cue condition). This main effect was qualified by a univariate group by target type interaction for RT, $F(2, 33) = 3.89$, $p < .05$. The PD group had an average RT that was 209.8ms faster for the Exogenous compared with the Endogenous Paradigm ($t = 3.06$, $p < .005$) (collapsing across cue condition). Similarly, the AD group had a RT that was, on average, 334.3ms faster for the Exogenous compared with the Endogenous Paradigm (814.6ms and 1148.9ms, respectively; $t = 4.53$, $p < .0001$). The elderly control group had a slightly faster RT in the Exogenous compared with the Endogenous Paradigm (586.2ms and 663.3ms, respectively) although this difference, in this analysis, was not significant ($t = 1.32$, $p = .19$).

There was no group by condition by target type interaction, $F(4, 67) = 0.94$, $p = .45$. Figure 7.6 illustrates that the AD group had slower RT than the elderly control and PD groups for both Exogenous and Endogenous Paradigms. The PD group had slower RT than the elderly controls for all conditions of the Endogenous Paradigm and all but the invalid condition of the Exogenous Paradigm (which were equivalent for the PD and the elderly control group).

A priori planned comparisons of the "costs plus benefits" for the PD group indicated that, for the Exogenous Paradigm, the "costs plus benefits" (88.1ms) was significant ($t = 2.96$, $p < .005$) while those for the Endogenous Paradigm (24.6ms) was not ($t = 0.82$, $p = .41$). Both the AD and the elderly control groups had significant "costs plus benefits" for both the Exogenous Paradigm (101.5ms and 226.1ms, respectively) and the Endogenous Paradigm (63.4ms and 225.4ms, respectively).

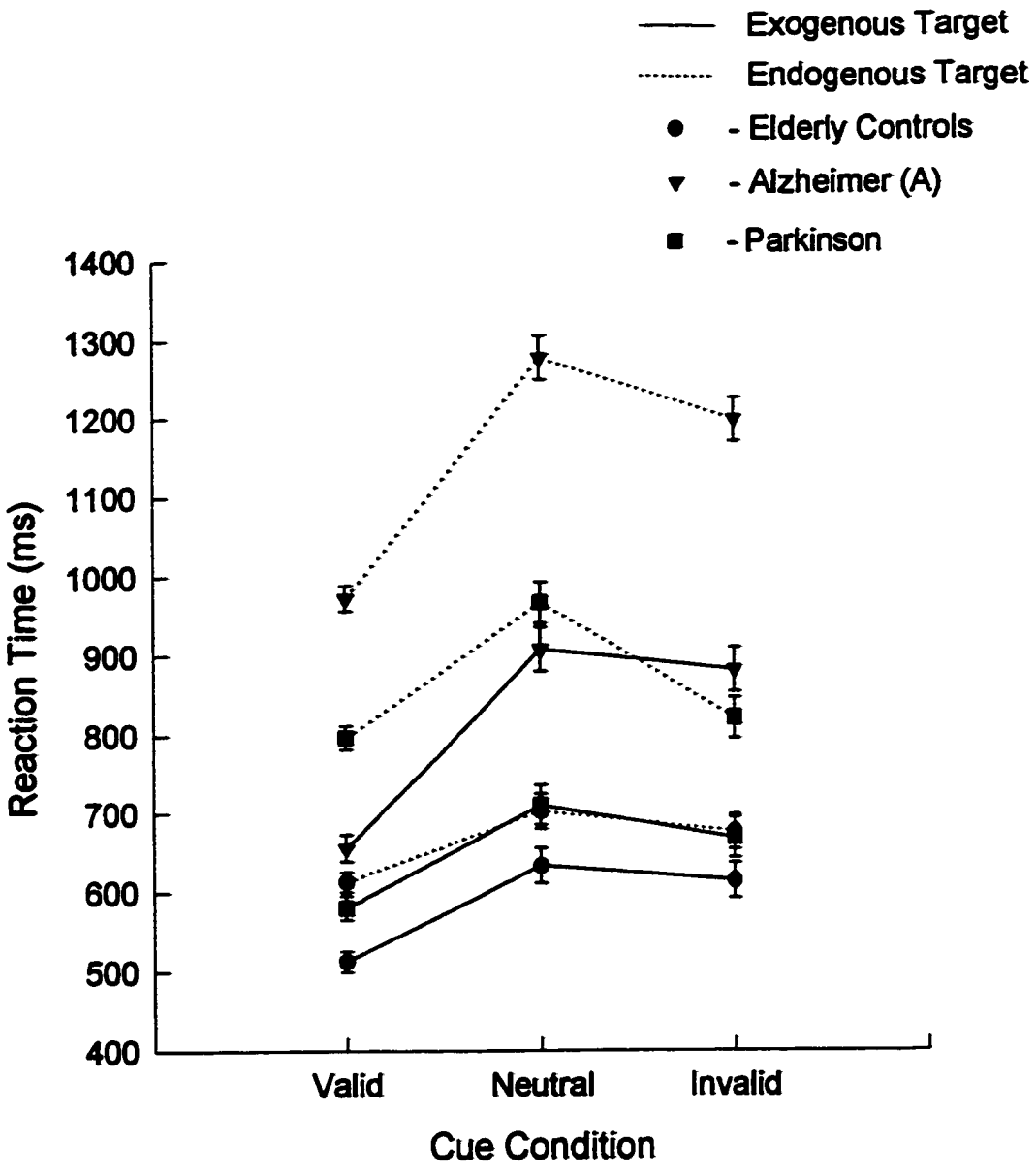


Figure 7.6 - Reaction time as a function of cue condition, target paradigm and group. The error bars represent the standard error of the least squares means.

Movement Duration Time (See Figure 7.7)

Results of the univariate mixed model ANOVA for MT revealed a marginal univariate main effect of group, $F(2, 33) = 2.50$, $p = .09$. The PD group had only marginally slower MT than the elderly control group (by approximately 58ms, $t = 1.69$, $p = .09$) and did not differ from the AD group (13.9ms, $t = 0.36$, $p = .72$) (collapsed across cue condition and target type). The AD and elderly groups differed only marginally ($t = 1.99$, $p = .05$), with the elderly subject group having a slightly shorter MT (72.1ms) than the AD group (again, collapsed across cue condition and target type). There was a univariate main effect of cue condition $F(2, 17) = 17.21$, $p < .0001$. The invalid condition had a significantly longer MT than the valid condition ($t = 5.53$, $p < .0001$) and only a marginally longer MT than the neutral condition ($t = 1.83$, $p = .07$). The valid condition had a shorter MT than the neutral condition ($t = 3.28$, $p < .001$). There was a univariate main effect of target type, $F(1, 33) = 8.06$, $p < .01$ with the Exogenous Target Paradigm having a shorter MT than the Endogenous Target Paradigm ($t = 2.82$, $p < .01$).

There was a univariate group by cue condition interaction, $F(4, 66) = 4.53$, $p < .005$, due to differing patterns of performance among the three groups across cue conditions. The PD group had significant differences between the invalid and the valid conditions (valid 37.9ms shorter than invalid, $t = 3.10$, $p < .005$) and the neutral and valid

conditions (valid 39.5ms shorter than neutral, t = 3.23, p < .005), but no significant differences between the invalid and neutral conditions (difference = 1.6ms, t = 0.11, p = .91). The AD group had significant differences between the invalid and valid conditions (valid 70.3ms shorter than invalid, t = 5.35, p < .0001) and the invalid and neutral conditions (neutral 51.5ms shorter than invalid, t = 3.20, p < .005) but no difference between the valid and neutral conditions (t = 1.44, p = .15). The magnitude of differences among the cue conditions was slightly greater for the AD than the PD group. For the elderly subject group, there were no differences among the three cue conditions (all p values > .05).

There was no group by target type interaction $F(2, 33)$ = 1.02, p = .37 and only a marginal group by condition by target type interaction, $F(4, 66)$ = 2.56, p = .05. This marginal interaction was due to differing patterns among the cue conditions for the three groups. For the Exogenous Target Paradigm, the elderly control and the AD groups had no significant differences among the cue conditions. However, in the Exogenous Target Paradigm, the PD had a marginally faster MT for the valid cue condition than the invalid (t = 1.73, p = .09) and the neutral conditions (t = 2.56, p < .05). In the Endogenous Paradigm, the elderly control group had no MT differences across cue conditions. The AD group had shorter MT for valid compared with invalid conditions (t = 6.97, p < .0001), and shorter MT for the neutral than the invalid

condition (t = 4.38, p < .05). The neutral and valid conditions were equivalent (t = 1.61, p = .11). For the PD group, in the Endogenous Paradigm, MT was also faster in the valid than invalid conditions (t = 2.98, p < .005). However, in contrast to the AD group, MT was faster for the valid than the neutral condition (t = 2.32, p < .05) and equivalent for the invalid and neutral conditions (t = 0.53, p = .60).

As Figure 7.7 illustrates, MT was shorter for the elderly controls than both the AD and PD groups in all conditions of both target paradigms. The AD and PD groups have equivalent MT with the exception of the invalid condition of the Endogenous Paradigm where the AD group have a longer MT.

A priori planned comparisons indicated that the PD group had only marginally significant MT "costs plus benefits" for the exogenous target type (27.8ms, t = 1.72, p =.09), but had significant "costs plus benefits" for the Endogenous Paradigm (48.0ms, t = 2.98, p < .005). For the elderly control group, there were no significant "costs plus benefits" for the Exogenous Paradigm (t = 1.01, p = .32) or the Endogenous Paradigm (t = 0.67, p = .51). For the AD group, there was no significant "costs plus benefits" for the Exogenous Paradigm (19.9ms, t = 1.13, p = .26) but there were for the Endogenous Paradigm (121.4ms, t = 6.97, p < .0001). Although the pattern of "costs plus benefits" was similar for the AD and PD groups, in the Endogenous Paradigm, the magnitude difference was much greater for the AD group.

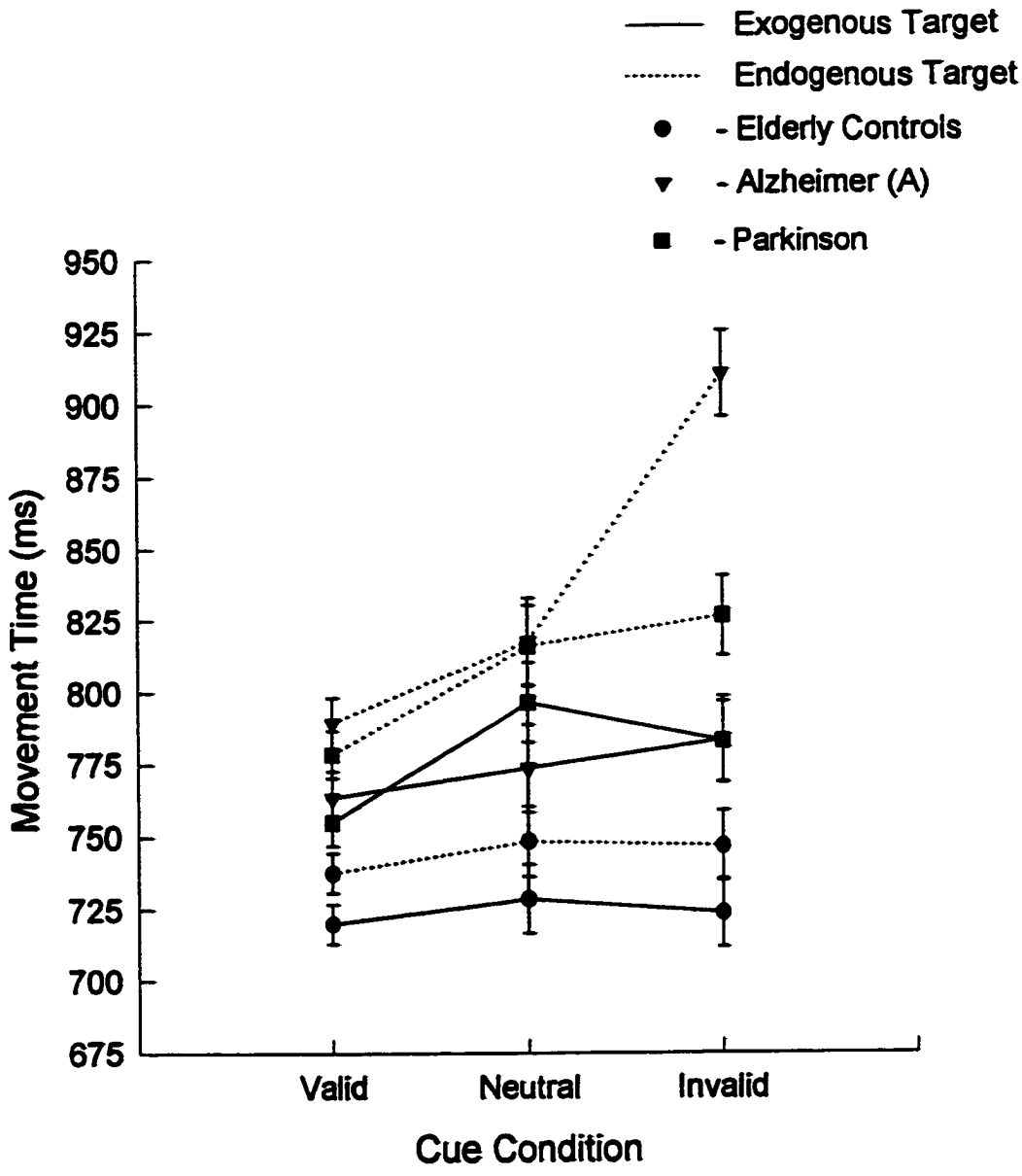


Figure 7.7 - Movement time as a function of cue condition, target paradigm and group. The error bars represent the standard error of the least squares means.

Peak Velocity (See Figure 7.8)

Results of the univariate mixed model ANOVA for PV indicated that there was a marginal univariate main effect of group, $F(2, 33) = 2.89$, $p = .06$. This was due to the PD group having lower PV (by an average of 9.2cm/sec) than the elderly control group ($t = 2.24$, $p < .05$) (collapsed across cue condition and target type). The PV was equivalent for the PD and AD groups ($t = 0.45$, $p = .65$) and the AD and elderly control groups ($t = 1.66$, $p = .11$).

There was no group by condition interaction, $F(4, 67) = 0.54$, $p = .70$, no group by target type interaction $F(2, 33) = 0.14$, $p = .87$, and no group by condition by target type interaction, $F(4, 68) = 1.37$, $p = .25$.

Similar to the elderly control group, a priori paired comparisons indicated that the "costs plus benefits" for the PD group was not significant for the Exogenous Paradigm (0.2 cm/sec, $t = 0.35$, $p = .73$) or the Endogenous Paradigm (0.5cm/sec, $t = 0.81$, $p = .42$). As noted earlier, the AD group had significant "costs plus benefits" for PV only in the Endogenous Paradigm ($t = 3.39$, $p < .001$) (due to their higher PV for valid cue conditions) and the elderly control group had no "costs plus benefits" for either target paradigm.

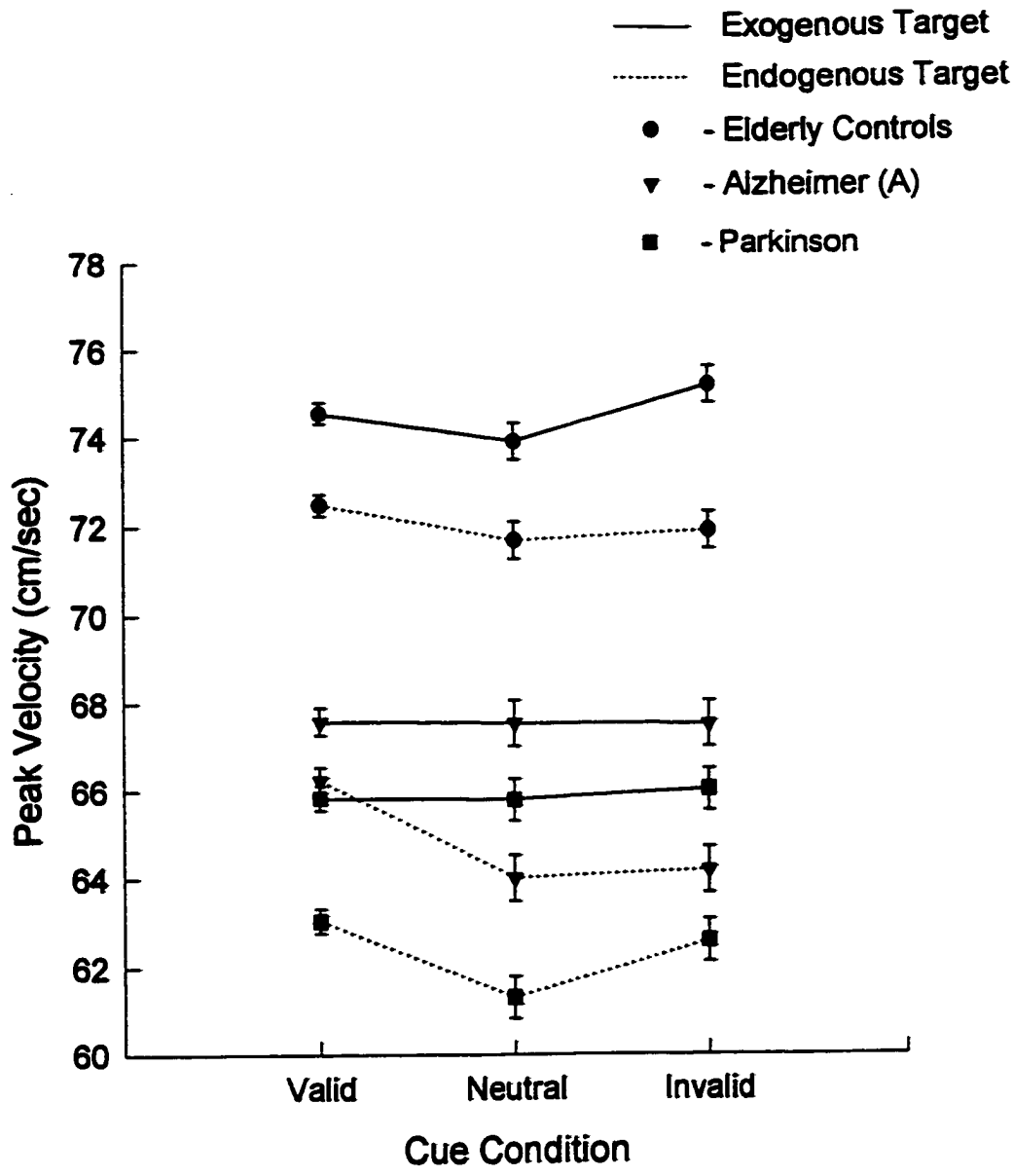


Figure 7.8 - Peak velocity as a function of cue condition, target paradigm and group. The error bars represent the standard error of the least squares means.

Percent Deceleration (See Figure 7.9)

Results of the univariate mixed model ANOVA for percent deceleration indicated that there was no main effect of group, $F(2, 33) = 1.33$, $p = .28$, no group by condition interaction, $F(4, 67) = 0.42$, $p = .79$, no group by target type interaction, $F(2, 33) = 1.19$, $p = .32$ and no group by condition by target type interaction, $F(4, 67) = 0.39$, $p = .82$. The average percent deceleration among groups and conditions ranged from 58% to 61%.

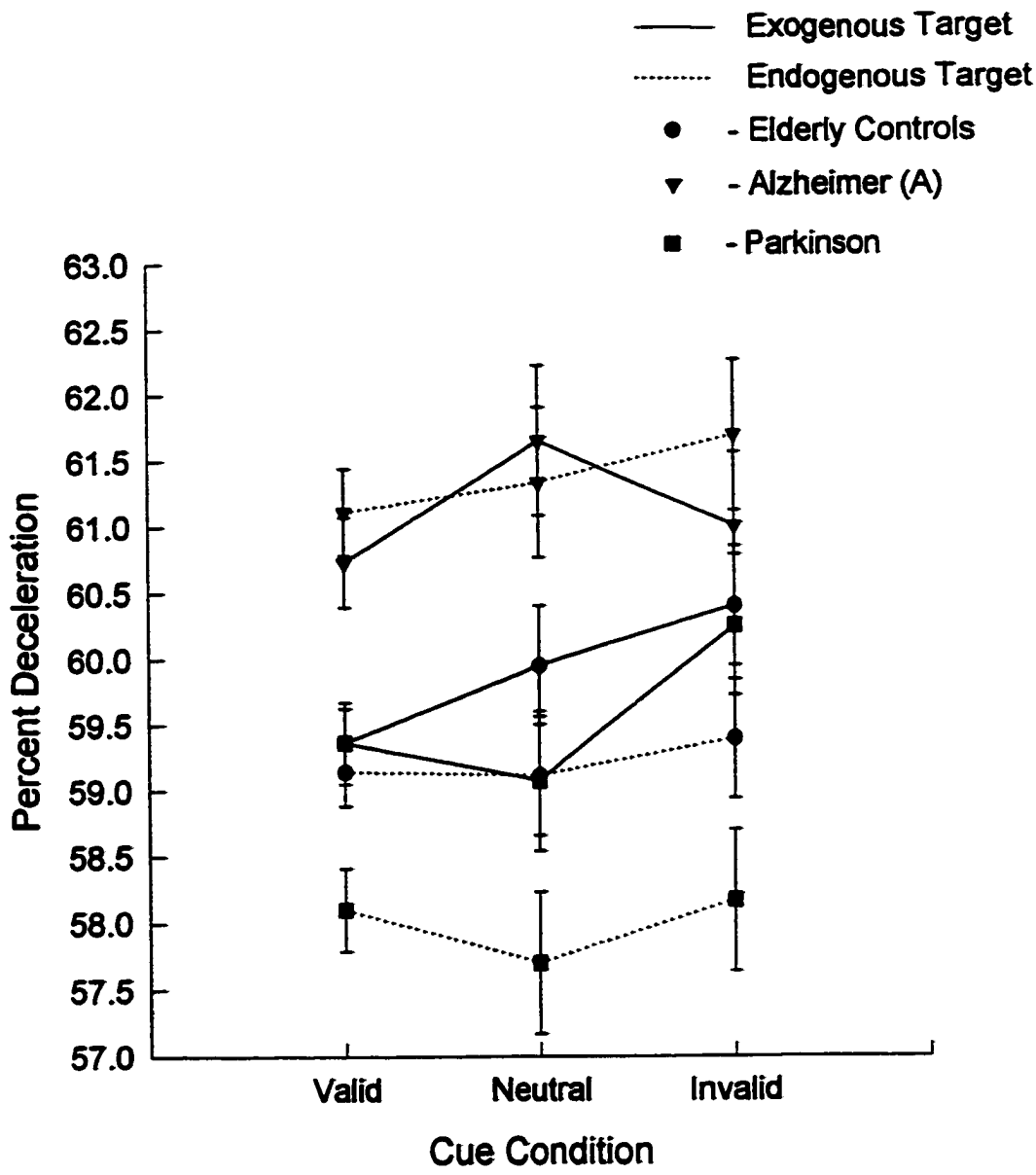


Figure 7.9 - Percent deceleration as a function of cue condition, target paradigm and group. The error bars represent the standard error of the least squares means.

Resultant Error (See Figure 7.10)

Results of the univariate mixed model ANOVA for resultant error indicated there was no main effect of group, $F(2, 33, = 2.01)$, $p = 0.15$, no group by condition interaction, $F(4, 70) = 0.73$, $p = .57$, no group by target type interaction, $F(2, 33) = 0.33$, $p = 0.74$, and no group by condition by target type interaction, $F(4, 70) = 0.90$, $p = .47$. Mean error for the PD group ranged from 9.6mm to 10.7mm for all subjects for both target types. For the AD group the mean error ranged from 7.7mm to 8.2mm for all subjects across both target paradigms. For the elderly control group, the mean error ranged from 8.1mm to 8.5mm. It is worth noting that, while the overall ANOVA was not significant (possibly due to a limited number of trials, resulting in low power for this analysis) post-hoc comparisons indicated that the PD group was less accurate than the elderly controls and the AD groups for all conditions of both target paradigms. The values are as follows: For the Exogenous Paradigm, the elderly subject group had smaller resultant error than the PD group in the valid condition (8.5mm vs 10.1mm, $t = 6.74$, $p < .0001$), the invalid condition (8.1mm vs 9.6mm, $t = 3.56$, $p < .001$), and the neutral condition (8.3mm vs 9.8mm, $t = 3.65$, $p < .005$). The AD group had a smaller resultant error than the PD group in the valid condition (8.2mm vs 10.1mm, $t = 6.99$, $p < .0001$), invalid (8.2mm vs 9.6mm, $t = 3.10$, $p < .005$), and the neutral condition, (8.0mm vs 9.8mm, $t = 3.92$, $p < .0005$).

For the Endogenous Paradigm, the elderly subject group had smaller resultant error than the PD group in the valid condition (8.4mm vs 10.4mm, $t = 8.03$, $p < .0001$), the neutral condition (8.5mm vs 9.6mm, $t = 2.42$, $p < .02$), and the invalid condition (8.4mm vs 10.7mm, $t = 5.47$, $p < .0001$). The AD group also had a smaller resultant error than the PD group (in the Endogenous Paradigm) in the valid condition (7.8mm vs 10.4mm, $t = 9.36$, $p < .0001$), the neutral condition (7.7mm vs 9.6mm, ($t = 3.84$, $p < .0005$) and the invalid condition (7.7mm vs 10.7mm, $t = 6.45$, $p < .0001$).

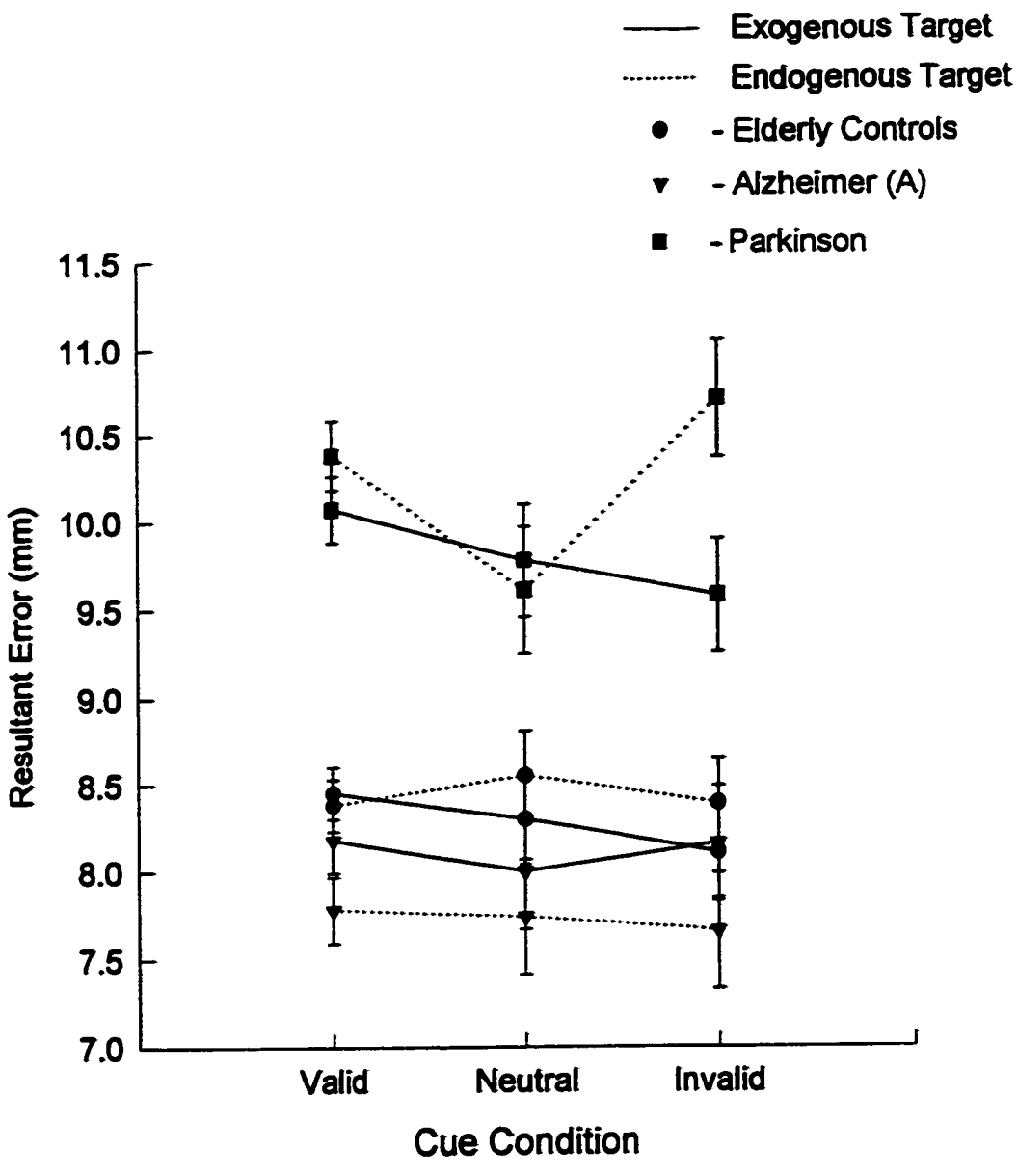


Figure 7.10 - Resultant error as a function of cue condition, target paradigm and group. The error bars represent the standard error of the least squares means.

Total Performance Time (RT plus MT) (See Figure 7.11)

Figure 7.11 illustrates the total time it took for a movement to be completed (from the time of target presentation) for each group in each of the target paradigms. That is to say the time it took to initiate the movement plus the time required to complete the movement after it had been initiated. This allows a qualitative view of differences within and among the groups for the two target paradigms.

In the Exogenous Paradigm, the total performance time for the PD group was approximately 8% longer for the neutral and invalid conditions and 12% longer for the valid conditions compared with the elderly controls (due to faster RT in the valid condition for the elderly controls). Compared with the AD group, the PD group had a total performance time of 15% less for the neutral and invalid conditions and 3% less for the valid conditions.

In the Endogenous Paradigm, the total performance time for the PD group was approximately 15% longer for the invalid condition, and approximately 20% longer for the neutral and valid conditions compared with the elderly controls. Compared with the AD group, the PD group had a total performance time of approximately 28% less for the invalid condition, 20% less for the neutral condition and 9% less for the valid condition.

Of the differences in total performance time among the three groups, the greater proportion was due to differences in RT as opposed to MT. As discussed in the previous chapter this

indicates that most of the processing and programming were completed prior to movement initiation.

In summary, the three groups differed least in the Exogenous Paradigm. As well, the AD subjects were more impaired than the PD subjects on total duration of the movement and particularly of proportionately longer RT's.

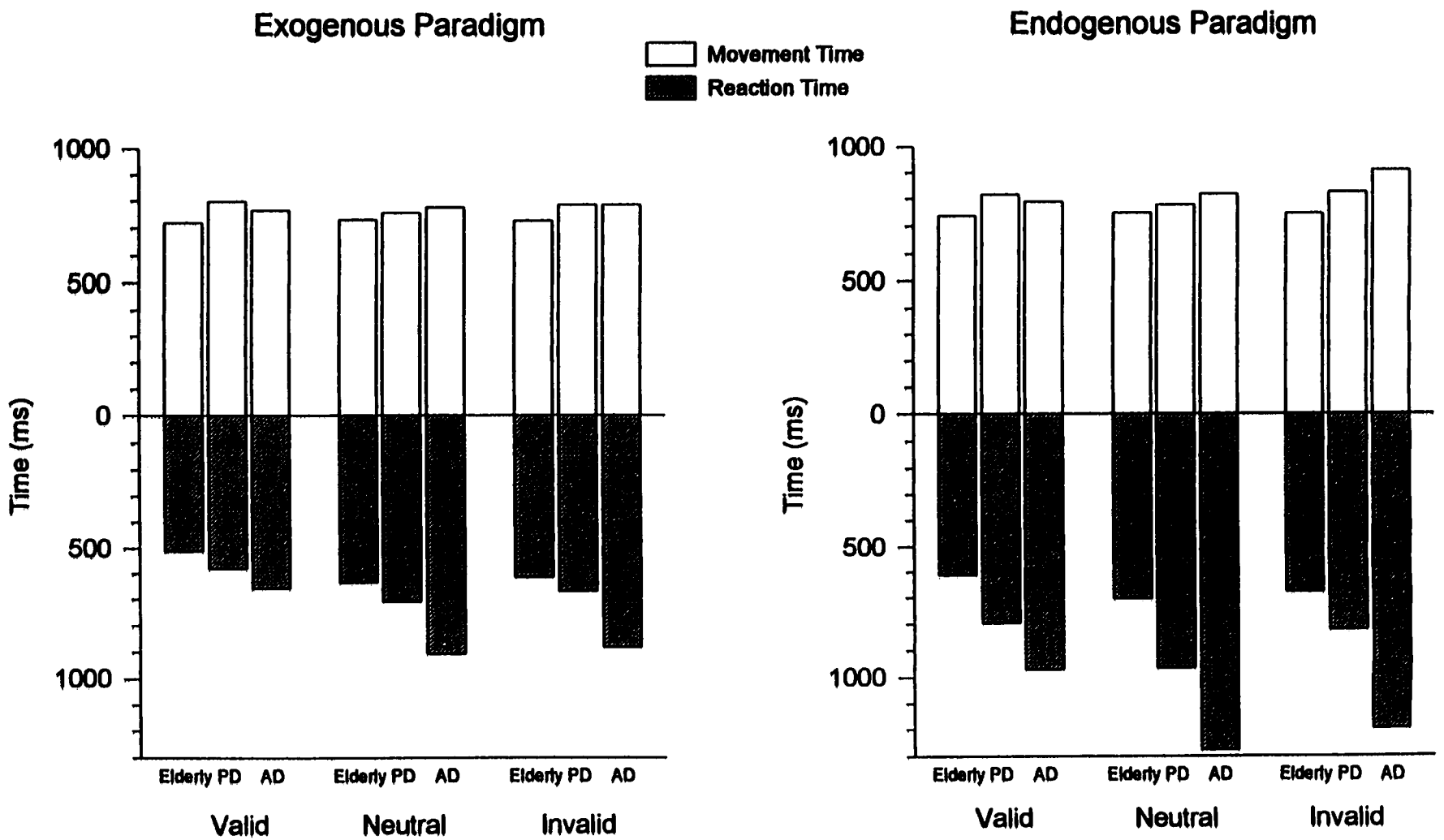


Figure 7.11 - Total Performance Time

DISCUSSION

The PD group had, overall, faster RT and a slightly higher PV for the Exogenous (compared with the Endogenous) Paradigm. Examining the "costs plus benefits" revealed that there were RT and MT "costs plus benefits" for the Exogenous Paradigm. In contrast, there was only "costs plus benefits" for MT in the Endogenous Paradigm.

The PD and the AD groups were impaired relative to the elderly controls on both the attentional and motor processes involved in the production of visually directed exogenous and endogenous overt movements. However, the pattern of impairment differed between the two clinical groups in both the Exogenous and the Endogenous Paradigms. As well, the PD group was generally less impaired than was the AD group.

The PD group had slower RT than the elderly control group in all conditions of the Endogenous Paradigm and in all but the invalid condition of the Exogenous Paradigm. This general slower RT for PD subjects is consistent with most studies which have noted slower RT for PD compared with control subjects (Brown et al., 1993; Caffarra et al., 1997; Jahanshahi et al., 1992).

The PD group, in the present study, also had faster RT in all conditions of both paradigms compared with the AD group. This is consistent with the results of Cossa et al. (1989), who found faster RT for non-demented PD subjects compared with AD subjects in a target discrimination and detection task.

Caffarra et al. (1997) reported no statistical difference in the mean RT values for AD and non-demented PD subjects although there was a pattern for the non-demented PD subjects in their study to have faster RT than the AD subjects. Given that in the Caffarra et al. (1997) study, RT was similar for the AD subjects and the PD subjects with dementia, it seems likely that the greater cognitive deficits of the AD group (i.e., lower MMSE scores), in the current study (compared with the PD group), may have contributed to their slower RT compared with the PD group. Thus, the cognitive deficits of the AD subjects, in this study, appeared to be associated with greater deficits in RT than were the motor deficits of the PD subjects. Wright et al. (1994) also reported faster mean RT values for PD compared with AD subjects but did not analyze the difference between the two groups statistically (which was approximately 77ms).

The PD group had longer MT than the elderly control group in all conditions of both paradigms. This is consistent with the results of previous research which found longer MT for PD subjects than controls (i.e., Jahanashahi et al., 1992; Muller & Stelmach, 1992; Praamstra et al., 1996). The PD group had equivalent MT to the AD group with the exception that the AD group had a longer MT than the PD group in the invalid cue condition of the Endogenous Paradigm. This appeared to be due to the particular difficulty of that condition for the AD group. To this author's knowledge, MT has not been previously

compared in these two patient groups.

PV was lower for the PD group than the elderly control group, consistent with previous results of other research (e.g., Inzelberg et al., 1990; Muller & Stelmach 1992). The finding of lower PV for PD subjects than elderly controls might be expected given that pathology of the basal ganglia is associated with deficits in the amount of force exerted and the rate of change of force (Yanagisawa, Fujimoto, & Tamaru, 1989). The PV of the PD group was equivalent to that of the AD group which, again, to this author's knowledge has not been investigated previously.

There was no difference in the proportion of the movement that was carried out in the deceleration phase among the PD, elderly control and AD groups. The deceleration phase is often thought to be the portion of the movement where error corrections are made. This would suggest that the PD group did not make any greater corrections during the final phase of the movement than did the elderly control or the AD subjects. However, although accuracy did not differ significantly among the three groups, there was a nonsignificant trend towards the PD group being less accurate than both the AD and elderly control groups. It is possible that, in the current study, the power to detect a significant difference was low. The PD group did maintain a constant accuracy across cue conditions, although, again, Figure 7.10 suggests a trend toward lower accuracy in the PD group for the invalid condition of the

Endogenous Paradigm.

The PD group showed an overall RT and MT advantage of the Exogenous over the Endogenous Paradigm. This result is consistent with those found for the elderly control and AD groups. This suggests that the advantage of exogenous overt orienting over endogenous overt orienting (sometimes interpreted as an automatic over controlled processing advantage) remains intact in both neurodegenerative disorders that were studied.

Consistent with the results of the elderly control and the AD groups, the PD group had significant RT "costs plus benefits" for the Exogenous Paradigm. This suggests that they were able to use cues to facilitate movements. The fact that they, like the elderly control and AD groups, had no "costs plus benefits" for any of the other dependent variables in this paradigm suggests that the PD group also made all necessary changes to an incorrectly preprogrammed exogenous overt movement prior to movement initiation. This result is similar to those of Jahanshahi et al. (1992) and Brown et al. (1993) who also noted all changes in reprogramming to take place prior to movement initiation for PD subjects.

In contrast to both the elderly control and the AD groups, however, the PD group had no RT "costs plus benefits" for the Endogenous Paradigm. Thus, it appears that the PD group did shift their attention and prepare a response to the cues in the Exogenous Paradigm (see Alternative #1, pg. 48).

While an examination of the RT measure suggests that PD subjects did not prepare a movement in response to the cues in the Endogenous Paradigm, when considering the MT "costs plus benefits" a somewhat different picture emerges. In contrast to their performance in the Exogenous Paradigm (where they had "costs plus benefits" for RT but not for MT), in the Endogenous Paradigm they had no "costs plus benefits" for RT but had significant "costs plus benefits" for MT (reversal of the exogenous pattern). This would suggest that the PD subjects did use the cues to prepare a response in the Endogenous Paradigm but that, if the cue was later determined to be invalid, they modified the planned response after the movement was initiated. The fact that the neutral cue condition had the highest RT of all the conditions in the Endogenous Paradigm adds further support for the suggestion that the PD subjects were using the informational cues to plan a response in the Endogenous Paradigm.

Thus, it appears that the PD group (similar to the elderly control and AD groups) were able to use cues to facilitate movement. For the PD group, this facilitation was greater in a situation where attention was shifted compared with situations where it was not. Further, when attention was shifted, PD subjects were able to complete all necessary changes prior to movement initiation. When attention was not shifted, PD subjects were unable to make necessary changes prior to movement initiation but had to do so during movement

execution. This result may indicate that the covert shift of visual attention was crucial for the PD subjects to gain an advantage of the valid precues prior to movement initiation. The results of Montgomery et al. (1991) may provide an explanation for the finding that the PD group was able to gain an advantage from the valid cues but that this advantage was confined to the movement execution phase for the Endogenous Paradigm. They suggested that there are two separate neural subsystems involved in motor control, one for movement onset (considered to involve the association cortex-caudate pathway) which affects RT (i.e., the decision "when" to move), and one for movement execution (considered to involve the somatomotor cortex-putamen pathway) which affects MT and velocity (i.e., the decision of "how" to move). The results of the Endogenous Paradigm of the current study would suggest that PD subjects are unable to use this association cortex-caudate pathway to effectively preprogram movement onset and take advantage of the valid cues. However, given that the results of the Exogenous Paradigm differed from those of the Endogenous Paradigm, this explanation would suggest that exogenous overt movements were programmed differently from endogenous overt movements, in PD. It appears that shifts of attention and/or exogenous stimuli in PD subjects may be necessary to facilitate the functioning of this association cortex. Another way to conceptualize this result for the Endogenous Paradigm in PD subjects (i.e., valid RT = invalid PT) is that it was

due, in part, to the endogenous target being under internal (voluntary) control (as opposed to the external or automatic control of the exogenous target). This would support the literature suggesting that individuals with PD have greater difficulty with internally (compared with externally) controlled movements (e.g., Brown et al., 1993; Kritikos et al., 1995; Majsaki, Kaminski, Gentile, & Flanagan, 1998). In this case, the "greater difficulty" would be evident by the lack of differences in RT for the valid and invalid cue conditions. It may also be that, although the cued information was not able to be used prior to movement initiation, some information about the cue was retained from the RT interval and was used during movement execution. It is possible that the PD subjects took longer to process the cued information in the Endogenous Paradigm (i.e.,internal condition) than in the Exogenous Paradigm (i.e., external condition), resulting in the information not being completely processed until the movement execution phase. The fact that the supplementary motor area is involved in the processing of internal cues and that this area is affected in PD, lends support to such an interpretation. Further, Morris and Iansek (1996) point out that the supplementary motor area generates the readiness potential and that in healthy individuals (with internally generated movements) the premovement supplementary motor area activation stops suddenly at movement onset to allow immediate movement initiation. In PD, however, they suggest that this

supplementary motor area activation is prolonged, resulting in a delay of the movement initiation. If one assumes that this delay is equivalent for valid and invalid conditions, this explanation would fit with the results of the current study finding no RT advantage for valid cues over invalid cues in the Endogenous Paradigm. The primary motor area is considered to be more involved in externally generated movements (i.e., the Exogenous Paradigm) and these movements have been noted to be less difficult for people with PD (for review see Cunnington, Bradshaw, & Iansek, 1996). This was the case in the current study where the PD subjects were able to show a RT advantage of valid over invalid cues in the Exogenous Paradigm.

The differences between the performance of the AD and PD groups in this study may be a reflection of the varying neuropathology between AD and PD. PD has been considered to mainly affect subcortical areas such as the basal ganglia, and involves the loss of dopaminergic neurons while AD has been considered to mainly affect cortical areas and involves deterioration of the cholinergic neuronal system and the formation of plaques and tangles (see Darvesh & Freedman, 1996 for a review). Consistent with this, the deficits of the AD subjects used in this study appeared to be more related to cortical-cortical interactions (i.e., frontal and parietal interactions) while those of the PD group appeared to be related more to cortical-subcortical interactions (i.e.,

supplementary motor cortex-basal ganglia interactions and primary motor cortex-basal ganglia interactions) that affected the mechanics of movements rather than the cognitive aspects of movements (i.e., executive functions).

For the Exogenous Paradigm, while the RT "costs plus benefits" for the PD group had a smaller absolute value than the elderly controls (88.1ms and 101.5ms, respectively), they did not differ statistically from those of the elderly control group ($F = 0.08$, $p = .78$). As previously described, the "costs plus benefits" was the only measure of cuing effects used in the current study (i.e., no separate "costs" were calculated; cf. Jonides & Mack, 1984). Since this measure was equivalent in the PD and elderly control group, this study did not provide evidence of for a deficit in the maintenance of attention in PD. This conclusion supports the research of Bennett et al., (1995) and Sharp (1990) who found no deficits in the maintenance of attention for PD subjects based on equivalent RT "costs" for PD and control subjects but is in contrast to the work of Wright and colleagues (Wright et al., 1990, 1994) who noted a maintenance of attention deficit in PD subjects based on the findings of reduced "costs" in PD subjects compared with controls. However, the "costs plus benefits" measure used in the current study is not directly comparable to the "costs" measure used in the previous studies.

The qualitative observations that were noted for the AD

subjects (i.e., using the wrong finger, more than one finger or the wrong hand when pointing to targets; pointing to the central target indicator rather than the actual target in the endogenous paradigm; being unable to take the hand down from the target after pointing to it; unable to lift the finger from the mouse) were not evident in the PD subjects, suggesting that these observed behaviors in the AD subjects were associated with their lower cognitive level and possibly were due to greater deficits in executive functioning.

The following chapter will summarize the relevant findings of this thesis for the four groups. Practical "everyday" implications of this work will be highlighted as will the advantages and limitations of the studies used in this thesis.

Chapter 8

GENERAL DISCUSSION

The results of the current study lead to some general conclusions about visual attention and motor programming in young adults, during the healthy aging process, and as a result of neurodegenerative disorders such as Parkinson's disease (considered to be mainly a motor disorder) and Alzheimer's disease (considered to be mainly a cognitive disorder). These conclusions will be presented in the following section.

Reaction time (RT) was fastest in healthy young adults and declined with the healthy aging process. There was a significant decline in RT with PD in comparison with healthy aging. There was a further decline with AD which became even worse with disease progression. Thus, while a disease that is known to produce motor deficits (PD) was shown to reduce RT, a disease that is associated with cognitive decline (AD) resulted in even more striking reductions in RT (than PD). That RT became much slower in those AD subjects who had more severe cognitive deficits (i.e., lower MMSE scores) suggests that greater degrees of cognitive deficits were associated with slower RT.

Movement duration (MT) was fastest for young subjects. It increased with healthy aging and increased further, and to a similar degree, with the diseases of AD and PD. Although both RT and MT became slower with healthy aging, AD and PD, the

magnitude of increase among the groups was greater for RT than MT. These results highlight the importance of cognitive processes in motor function, particularly in RT.

Peak velocity (PV) was highest for young subjects, and lowered similarly with heathy aging, AD and PD. Constant accuracy appeared to be the goal of both exogenous and endogenous overt movements. However, it appears that PD (in the current study) was associated with a decrease in accuracy.

Exogenous overt orienting had an advantage over endogenous overt orienting for the speed of planning and executing a visually directed pointing movement for all groups used in this study. This indicates that the advantage of movements to targets that elicit exogenous overt orienting (i.e., externally processed cues, or automatic processing), was maintained during the healthy aging process, and with the diseases of AD and PD. Since it is possible that the young adults and the AD subjects used in this thesis may not have shifted attention in response to the cues in the Exogenous Paradigm, this exogenous advantage cannot be solely attributed to a covert shift of visual attention. More convincing evidence for this conclusion comes from the observation that the exogenous/endogenous difference in RT is there even in the neutral condition (where there was assumed to be no shift of attention or motor programming). It seems likely that this exogenous advantage may result from the fewer requirements of the decision process in the Exogenous compared with the

Endogenous Paradigm.

Visual precues (i.e., advance target information) were used to facilitate movements for all groups. This was indicated by the advantage of movements in valid conditions compared with that of invalid cue conditions. The facilitation produced by the precues differed (with regards to which dependent variables were affected by the cues and how they were affected) as a function of target type and group. Healthy elderly adults as well as those with AD and PD appeared to make all changes to an incorrectly preplanned exogenous overt movement prior to movement initiation, since invalid cues led to increased RT but not MT and were not associated with any changes in the other dependent measures. However, these changes, prior to movement initiation, took significantly longer for the AD subjects than for the healthy elderly subjects. In contrast to the elderly controls and the AD groups (in the Exogenous Paradigm), the young subject group made some changes prior to movement initiation but made further adjustments during movement execution (increased RT plus increased MT for invalid cue conditions). This ability (to make changes both prior to movement initiation and during movement execution), for the young subjects, allowed them to initiate and thus complete movements with greater speed while maintaining equivalent accuracy to the other groups.

It was in the Endogenous Paradigm that the groups used in this study differed most. For endogenous overt movements,

healthy elderly individuals, once again, made all changes to an incorrectly preplanned movement prior to movement initiation while the healthy young individuals made some changes prior to movement initiation and further adjustments during movement execution. It is possible that this was due to the elderly control group adopting a more cautious approach to making movements (i.e., not to begin the movement until all aspects of the movement were clearly and correctly programed) compared with the young group. Thus, in some respects, subjects with mild AD exhibited a pattern similar to that of the young healthy subjects (in the Endogenous Paradigm) since they also made some changes prior to movement initiation and further adjustments during movement execution. However, because the AD subjects were much less efficient in making their movements than the healthy young subjects (i.e., greater RT and MT than the young group), it appears that making adjustments during movement execution was more a matter of underestimating the adjustments required to the program that they had reconstructed during the RT interval than a matter of employing an efficient response strategy which maximized the speeded processing of information. Clearly, the mild AD group had particular difficulty adapting to unexpected changes in the target position (i.e., invalid cue condition), particularly if they had to make changes to a motor program without having an external event to "pull" their attention to the new, correct target position (i.e., the Endogenous

Paradigm). Although the PD group appeared to rely completely on modifications to the planned motor response during its execution, they appeared to be much better able to respond to changes in the expected target location in the Endogenous Paradigm relative to the AD group.

The more impaired AD group [i.e., AD(B)] were unable to perform the endogenous overt movement task at all, and, in exogenous overt movements, they were found to make changes during movement execution (i.e., increased MT) in addition to those made prior to movement initiation. This was in contrast to the milder AD group [AD(A)] who made all changes in the RT period. Thus, while still only mildly impaired, on the basis of standard screening for cognitive status, the more advanced AD subjects used in this study were impaired even with exogenous targets, suggesting that they had significant difficulty adapting to unexpected events (i.e., invalid cue condition) even if there were external events to draw their attention. Thus, these results suggest the possibility that there is a hierarchy of decline in AD such that exogenous or automatic processing is fairly well preserved in the early stage of the disease while endogenous or controlled processing begins to decline at that time. As the cognitive decline continues with disease progression, the ability to use exogenous or automatic processes also undergoes a decline.

The performance of subjects in the PD group (for endogenous overt movements) was in contrast to all other

groups in that they made all changes to an incorrectly preplanned movement during movement execution (MT) with no changes being evident prior to movement initiation (RT). Thus, for endogenous overt movements, they appeared to have an equivalent advantage for movement initiation whether they were required to move to an expected or to an unexpected location, but had an added disadvantage for the duration of the movement when required to move to an unexpected location. Thus, a covert shift of attention, such as was possible in the Exogenous Paradigm, appeared to be necessary for the PD subjects to gain an advantage in the initiation of movements to validly cued targets.

Table 8.1 illustrates the "costs plus benefits" for RT and MT (which are the variables that provide the most relevant information) for all groups.

A comparison of the RT "costs plus benefits" between the Exogenous and Endogenous Paradigms for each group suggests that young healthy individuals may not have shifted their attention to the cue but merely planned a response (i.e., marginally smaller "costs plus benefits" for the Exogenous versus the Endogenous Paradigm; see Alternative #3, pg. 49). Similarly, the AD(A) group had equivalent RT "costs plus benefits" for the Exogenous and Endogenous Paradigms. This suggests that, they too, may not have shifted their attention in response to the cues in the Exogenous Paradigm but merely prepared a response (see Alternative #3, pg. 49). It is also

possible that they did not shift attention or program a movement prior to the target presentation (see Alternatives #7, pg. 51 and #6, pg. 54). However, unlike the young subject group (who made some changes to the movement program in the RT interval and further changes during the MT period), the AD(A) group made all changes to the movement program (in the Exogenous Paradigm) prior to movement initiation. As well, the AD group took much longer to make changes to their movement program than did young subjects. Those AD subjects with more severe cognitive deficits [AD(B)] were unable to carry out endogenous overt movements.

In contrast to the young and the AD(A) groups, the healthy elderly group appeared to have shifted attention in response to the cues (in the Exogenous Paradigm) and received an extra advantage of having done so in addition to having planned a movement (see Alternative #1, pg. 48) compared to having planned a movement only (see Alternative #3, pg. 49). Support for this can be seen in their faster RT to valid cues compared with invalid cues in the Exogenous Paradigm compared with the Endogenous Paradigm (refer to Figure 5.1).

Those with PD showed a strong RT advantage for movements with a covert shift of attention in addition to motor programming (Exogenous Paradigm) compared with motor programming only (Endogenous Paradigm), which showed no advantage of RT.

Table 8.1

"Costs Plus Benefits" for RT and MT for all Subject Groups for both Paradigms.

Exogenous Paradigm

Group	RT	MT
Young	55.3ms ($p < .0001$)	12.3ms ($p < .01$)
Elderly	101.5ms ($p < .0001$)	3.8ms (ns)
AD(A)	226.1ms ($p < .0002$)	19.8ms (ns)
AD(B)	298.7ms ($p < .001$)	121.4ms ($p < .0001$)
PD	88.1ms ($p < .01$)	27.8ms ($p < .01$)

Endogenous Paradigm

Group	RT	MT
Young	74.3ms ($p < .0001$)	29.7ms ($p < .0001$)
Elderly	63.4ms ($p < .0001$)	9.2ms (ns)
AD(A)	225.4ms ($p < .0002$)	121.4ms ($p < .001$)
PD	24.6ms (ns)	48.0ms ($p < .0001$)

When examining the total performance time (i.e., RT plus MT) of the groups, it is clear that the AD subjects were, overall, the slowest of all groups to initiate and complete movements (i.e., RT plus MT). The AD(A) group took over two seconds to complete movements to invalidly cued targets in the endogenous target paradigm. One might have considered it more likely that the PD group would take the longest for movements since PD is most often considered to be a "movement disorder". As well, the AD group had the greatest proportional increase in RT compared with that of MT for any of the groups. This indicates that they were more impaired on the processes which take place in the RT interval than were the other groups used in this study. Results of this thesis, then, suggest that the cognitive impairments associated with AD are easily apparent in the production of relatively simple movements. Perhaps, not surprisingly, these deficits are more evident for endogenous overt movements (i.e., controlled processes) than for exogenous overt movements (i.e., automatic processes) and there may be a hierarchy of decline in these abilities that parallel the decline in cognition in AD. Interestingly, the deficits in movement production and completion are less impaired in PD than in AD, again, pointing to the importance of cognition in motor function. However, PD subjects had a pattern of performance that was distinct from that of healthy aging or of AD and this was particularly true of endogenous overt movements.

Practical "Everyday" Implications

The findings of this thesis have numerous potential practical "everyday" implications for the healthy elderly and for the clinical groups used in this study. In the following section, these issues are discussed, particularly in relation to driving privileges, a prominent social concern and one that has been the source of considerable debate.

Driving is a particularly relevant task to use as an example since the effects of impaired visual-motor behavior can easily be appreciated. In addition, it is a difficult issue to address for families and physicians of those who are elderly, and/or who have AD and PD as well as being difficult for the patient themselves. In part, this issue is contentious because driving is such an important behavior in North American Society, providing people with independence (Dubinsky, Gray, Husted, Busenbark, Overfield, Wiltfong, Parrish, & Koller, 1991; Duchek, Hunt, Ball, Buckles, & Morris, 1997) that can be related to their sense of self-worth.

The findings of this thesis have suggested that healthy aging results in slower RT, longer MT, and lower PV. It has also illustrated that healthy elderly subjects are more apt to make any necessary changes to a previously planned motor response prior to beginning to move (which increases their RT considerably compared with young adults). This slowing of RT, combined with an overall slowing of movement execution, has

implications for daily activities, particularly for situations requiring them to react quickly to unexpected events. This may make them at greater risk for accidents and injuries in situations such as driving, which may require the rapid production of a motor response to an unexpected event. In fact, this greater risk has been documented. For example, Dobbs (1997a) points out that when exposure to driving is considered, the crash rate of elderly drivers (over age 70) is equivalent to, or greater than, that of the "highest-risk" group (i.e., 16-24 year old drivers). Owsley, Ball, Sloane, Roenker, and Bruni (1991) have developed a model in which they concluded that degree of visual attention (described as "peripheral visual awareness", analogous to endogenous covert orienting of attention) was the strongest predictor of motor vehicle accidents in the elderly. Further, they point out that most accidents related to deficits in visual attention (in their study) occurred at intersections, which, Dubinsky et al. (1997) suggests, is where the majority of the critical decisions in driving must be made. In addition to accidents at intersections, Lerner (1994) found that elderly individuals are over-represented in accidents where they fail to yield the right-of-way (Lerner, 1994). Lerner (1994) attributes this to a slowing of RT and suggests the need to adapt the road environment to ensure that older drivers have sufficient time to react to environmental events.

AD further complicates the issue of driving and is

currently a major social concern. This concern was highlighted in an international symposium titled "Alzheimer's Disease and driving: Current research and public policy" which was held in 1996. Numerous studies have found that those with AD have increased rates of accidents compared with elderly controls when equated for number of miles travelled (Dubinsky et al., 1997; Friedland, Koss, Kumar, Gaine, Metzler, Haxby, & Moore, 1988). Friedland et al. (1988) reported that the majority of the accidents for AD subjects occurred at intersections, traffic signals, or while changing lanes. Dobbs, (1997a, b) using actual driving road tests (with driving instructors and in a car equipped with dual brakes), identified a specific group of driving error types which distinguished individuals with AD from healthy elderly adults. These included: going the wrong way on a freeway, and stopping at a green light. Perhaps in these situations the environmental cues set up an expected pattern of behavior (e.g., "stop at the lights") that could not be overridden when events changed (e.g., the light changes from red to green).

An example of how deficits in visual-motor integration would be hazardous during driving was presented in a single-case study of an 80 year old man with mild to moderate AD. He had four accidents during the course of an 18 mile drive using a driving simulator (Reinach, Rizzo, & McGehee, 1997). The first accident resulted from the driver steering into the left lane to pass a truck and not braking until 0.2 seconds before

crashing into a vehicle that was already in the left lane. The second accident occurred at an intersection where a vehicle was stopped with brake lights and left turn signal on. The subject was going over 80mph during the 5 seconds prior to crashing into the rear of this stopped car and never released the accelerator or touched the brakes, although he was noted to have had his eyes on the road for at least 3 seconds prior to crash. The third accident resulted when the driver encroached on a slow moving car but did not begin to release the accelerator until less than 1 second prior to crashing into the car and never touched the brake. The forth accident occurred when the driver released the accelerator only 1 second prior to crashing into a vehicle that was slowing and had put on the left turn signal at an intersection.

Despite the fact that such driving difficulties have been noted in individuals with AD, these individuals are often able to pass a standard driving test. Parasuraman and Nestor (1992) suggest that this is because most driving skills are "automatic" and the result of procedural knowledge, usually spared in the early stages of AD (consistent with the results of this thesis). As well, individuals with AD may benefit from environmental cuing of other drivers on the road such as stopping at a red light/stop sign because the driver in front does (Hunt, Murphy, Carr, Duchek, Buckles, & Morris, 1997) which may aid them during a driving test. Hunt et al. (1997) pointed out that these behaviors are consistent with anecdotal

reports from caregivers who say the AD drivers follow the flow of traffic of other drivers (e.g., slow down or stop at intersections with green lights on to wait and see how other drivers were proceeding before they advanced) but may fail to use the additional cues from the other traffic (e.g., follow another car on a left turn through the intersection but fail to take note of oncoming traffic, making the turn unsafe). The results of the current thesis would provide support for the suggestion that persons in the early stages of AD are more dependent on the availability of external cues in the environment and are able to benefit from such cues in order to produce behaviors that do not differ greatly from healthy controls. However, the great difficulty that the AD subjects had with endogenous cues (in the current study), and their large increase in RT (compared with healthy elderly subjects), make them at high risk for incidents in which avoiding an accident depends upon an internally generated modification of an anticipated motor response.

Consistent with the findings of this thesis, where the more severely cognitively impaired AD subjects demonstrated significant problems even when external cues were available to facilitate movement reprogramming, Drachman and Swearer (1993) noted that vehicle crashes increased with each year of driving after diagnosis of AD.

There is only minimal literature on driving and PD. Nonetheless, a study by Dubinsky et al. (1991) examined

driving in PD and control subjects and found that, over a three year period, the number of accidents per million miles of driving were significantly greater for those PD subjects with higher Hoehn and Yahr scores. There was an average of 10 accidents per million miles of driving for those with Stage 1, an average of 50 for those with Stage II, and an average of 80 for those with Stage III. No details on types or locations of accidents were provided. Dubinsky et al. (1991) attributed the increase in number of accidents with the increase in Hoehn and Yahr scores to increased difficulties in carrying out preprogrammed movements and with the reprogramming of motor activities. While the current thesis did not have a sufficient number of subjects to break the PD group down by Hoehn and Yahr staging, it was evident that those with PD also had difficulty with the initiation of movements that had to be generated internally (i.e., in the absence of an external event to first draw their attention). The increase in RT, in this situation, was much greater than that of healthy elderly individuals, and, although not as impaired as the AD group, the PD patients could be expected to have increased risks for accidents also. It is possible that, those with PD who do not have significant cognitive impairment, would be able to compensate for this deficit by beginning to make necessary changes to motor programs greatly in advance of the time when the program would have to be executed to completion (e.g., begin braking at a greater distance before an intersection

than a healthy person would generally do). However, this type of compensatory strategy would be unlikely to be helpful in situations where immediate action is necessary (e.g., car suddenly pulls out in front of a person) or under conditions in which changes to an unexpected event are under endogenous control.

While it has been stated that the most common reason for accidents is "driver inattention" (Duchek et al., 1997), results of this thesis suggest that such "inattention" may be a rather complex set of issues. The driving errors described previously for all groups are examples of events in the environment that necessitate a reevaluation of the movement program that is currently being conducted, making changes to this program, and initiating and executing the new program. These environmental events may be either exogenous (e.g., the car in front quickly stopping or slowing) or endogenous (e.g., making a decision to change lanes and pass a slow-moving car). In this thesis, the experimental setting provided minimal visual and auditory distractions, ample instructions, rest periods, and self-paced timing of the trials. Since the healthy elderly controls performed more slowly on the exogenous and endogenous movement tasks compared with young adults, and the PD and AD groups were impaired relative to elderly controls, one could anticipate that such difficulties would be much greater in a driving situation where there can be many visual and auditory distractions, and movements must

be initiated and executed with speed and accuracy.

The fact that driving is a highly visible topic in society (and in the health care profession) currently, does not preclude the importance of other issues relating to visual and motor integration in daily living. Amrhein, von dras, and Anderson (1993) suggest that difficulties with motor programming in the elderly may predispose them to serious falls (a prominent concern for health care providers of the elderly), and cause deficits in any human-machine interaction. One would expect this situation to be exacerbated for those with AD. Indeed, Oleske, Wilson, Bernard, Evans, and Terman (1995), examining a sample of community-dwelling AD patients, determined that falls (usually from stairs) were the most common accident reported, followed closely by striking portions of their body against stationary objects such as furniture or building structures. Those with greater levels of cognitive deficit reported a greater incidence of these types of accidents. Such findings would be consistent with the visual-motor integration deficits noted in this thesis as, presumably, these types of accidents result from inefficient programming and reprogramming of endogenous overt movements.

While PD subjects (without the severity of cognitive impairment of those with AD) may have less difficulty with such situations, they could be expected to have some similar problems to AD patients as they also appear to have to rely heavily on external events to facilitate movements and have

greater difficulty with self-generated movements. Of particular importance for PD subjects is the finding in this thesis, that even when endogenous environmental cues are available, PD subjects are unable to make changes to a movement prior to movement initiation. Rather, they must rely on being able to make the required changes during movement execution. This would place them at particular safety risk in numerous situations.

While issues of driving accidents and accidental falls have been associated in the literature with deficits in visuomotor integration, there are many common activities of daily living that the average healthy individual may take for granted that those with AD and PD will have difficulties with, particularly with disease progression. Some examples include: operating appliances such as stoves, coffee makers, using a sewing machine or other equipment for hobbies (e.g., saws and tools for woodworking), or programming the alarm system for the home. While difficulties with some of these activities may merely be irritating for the individual (e.g., slowness or difficulty programming the alarm system), others may challenge the safety of the individual in the home (e.g., using tools). As well, in everyday "normal" situations these difficulties may not be noticeable (as the person carries out "automatic" tasks). More likely, it will be in a novel or emergency situation that they will become issues (e.g., something on the stove catches afire; the electric saw slips from the hand)

when the requirement for fast accurate movement is imperative to prevent injury. The results of this thesis suggest that these activities may not produce equivalent levels of difficulty for the healthy elderly (as for those with AD or PD) but, nonetheless, may take longer for the healthy elderly to perform efficiently relative to healthy young adults.

Scope Of This Thesis

As is the case with most research, the paradigms used in this thesis have both advantages and limitations.

Advantages

The results of the studies conducted in this thesis point to the importance of examining the kinematic aspects of movements in addition to RT. The results also suggest the importance of integrating the cognitive (i.e., attention) and the motor (i.e., programming) literature in elucidating subtle differences in attentional and motor processes that occur with healthy aging and among clinical groups such as the AD and PD subjects used in these studies.

To illustrate the importance of integrating the kinematic variables with RT, one can examine what major results would not have been available had this work used only RT as a dependent measure. For example, one would not have realized that the young subjects made some changes to preplanned motor programs prior to movement initiation but made further adjustments during movement execution (for both exogenous and endogenous overt movements). In the same vein, one would have

missed the fact that the elderly, in contrast to the young, made no "on-line" adjustments to either exogenous or endogenous overt movements but, rather, made all necessary changes prior to movement initiation (resulting in longer RT but no change in MT). This would have made the performance of elderly controls look worse than they actually are (compared with young subjects). That the AD(A) and the PD groups, while having a similar pattern to the elderly control subjects for exogenous overt movements, differed from controls (and from each other) in the movement kinematics of endogenous overt movements would not have been noted (with the exception of reduced RT "costs plus benefits" compared with the elderly controls). As well, the fact that the AD(B) group had kinematic characteristics for exogenous overt movements that resembled those of the AD(A) group for endogenous overt movements would not have been evident. The differences in the main effects of the kinematic variables between the groups and between the two experimental paradigms would also have been unavailable.

Integrating the cognitive (i.e., Exogenous Paradigm) and motor (i.e., Endogenous Paradigm) perspectives with the kinematic perspective allowed for the examination of the integration of shifting of attention and aiming movements in a manner that had practical "everyday" implications (since reaching to objects is a key part of human daily functioning). Those researchers whose interest lies in examining attention

or motor functioning from a more purely theoretical perspective may find the paradigms used in this thesis somewhat limited because the processes were less "pure". Undoubtably, this integration of the attentional and motor perspectives has made the results much more challenging to interpret than may have been the case if the results were examined from only one of those perspectives in isolation. However, this is also the strength of the current work. It illustrates the complexity of the integration of visual attention and motor programming in the execution of movements in human daily activities. The fact that the motor task used in this thesis was a very simple one (i.e., pointing to and touching a location with the index finger) and that it was able to identify deficits associated with healthy aging and neurodegenerative diseases and to elucidate differences between two clinical groups, suggests that the difficulties in visual-motor integration in everyday living for these groups are likely equally dramatic as those noted in this thesis if measured appropriately.

The importance of using endogenous overt orienting (or controlled processing) was critical to this thesis in examining differences in attention and motor functioning among clinical groups. It was this paradigm which revealed the greatest differences among the groups (compared with the exogenous paradigm). This is consistent with research that suggests that the exogenous (automatic) processes remain

intact for a longer time than the endogenous (controlled) processes in some neurodegenerative disorders.

In the shift of attention literature, generally, endogenous cues are paired with exogenous targets. As well, even in the motor programming literature (and often in the kinematic literature) the targets are usually exogenous in nature (in that instruction as to where to move is provided by the target positions lighting up). In the kinematic literature, precues have not generally been incorporated into the experimental paradigm. Again, the current thesis had an advantage by pairing the Exogenous and the Endogenous Paradigms.

A further advantage of this thesis was its comparison of non-demented PD subjects with AD subjects (rather than PD subjects with dementia, which are often compared with AD subjects in the literature). This allowed for a more clear separation, examination, and comparison of cognitive deficits and motor deficits associated with neurodegenerative processes and their effects on the processes of covert shifts of attention and motor programming.

Limitations

One limitation of this work that has been touched on briefly in many of the chapters involves the comparisons made between the Exogenous and the Endogenous Paradigms. As previously described, the purpose of this work was to examine the effects of covert shifts of visual attention on motor

programming. The Exogenous Paradigm (i.e., endogenous cue followed by exogenously presented target) was deemed to be insufficient to examine this question as the endogenous cue provided information about the location to shift attention to and about the movement to be preprogrammed. To attempt to separate these two effects, the Endogenous Paradigm (i.e., endogenous cue followed by endogenously presented target) was developed. This paradigm was considered to isolate motor programming from a covert shift of visual attention by removing the advantage of shifting attention to a peripheral target location (i.e., by presenting the target information at centre). However, direct comparisons between the two paradigms remained problematic since the actual movements continued to differ with regard to the requirements for a shift of attention (i.e., overt exogenous vs overt endogenous). This made it difficult to distinguish whether the effects noted in the exogenous paradigm were due to covert endogenous shifts of visual attention that preceded the movement, or to processes elicited by the abrupt onset target.

One might also suggest that in the Endogenous Paradigm, the subjects could have shifted their attention to the periphery at the presentation of the cue and shifted it back to centre again when the target was presented. While there is no way to conclusively prove that this did not happen, it seems unlikely given the results. For example, the healthy elderly subjects had greater RT "costs plus benefits" for the

Exogenous compared with the Endogenous Paradigm, presumably due to the advantage of shifting attention in the valid condition. In contrast, the young subjects had only marginally smaller RT "costs plus benefits" for the Exogenous compared with the Endogenous Paradigm, presumably due to no shift of attention in the Exogenous Paradigm. As well, the PD subjects had "costs plus benefits" for the Exogenous Paradigm but none for the Endogenous Paradigm, suggesting a strong advantage for covert shifts of attention in addition to motor programming.

The analysis examining the "meridian effect" had low power to find effects because it used only invalid trials and was further broken down by hemispace of target.

A greater number of valid and invalid trials may have increased the power to find differences in the "costs plus benefits" between the two experimental paradigms for each of the groups and for the differences in "costs plus benefits" between the groups for each of the experimental paradigms. However, given that (in three of the experimental groups) the subjects used were either elderly and/or had neurodegenerative diseases, the effects of subject fatigue precluded the collection of a larger number of trials. Given that the neutral condition did not appear to be truly "neutral" (i.e., it did not fall between the valid and invalid trials for the dependent variables, which is required for it to be a baseline, or neutral, measure), future research might consider eliminating the neutral trials in order to increase the number

of valid and invalid trials as a way in which to increase the power of the analyses. In the current study, this was not an option, as the neutral trial issue did not become evident until after data collection and analyses were conducted.

A further limitation caused by the "non-neutrality" of the neutral cue was that this precluded the analysis of separate "benefits" (i.e., neutral minus valid) and "costs" (i.e., invalid minus neutral) which have traditionally been calculated in the orienting of attention literature to assess the initial "shift of attention" and the "disengagement of attention", respectively.

The use of resultant error rather than constant and variable error may have limited the information gained about the end point of the movement across groups (i.e., an inability to examine undershoots or overshoots of the target). This was not considered prior to the study and, in retrospect, was unable to be calculated due to an equipment error that did not allow the consistent calculation of the x, y and z coordinates directions.

Another limitation involves the sample sizes and representativeness. The studies conducted in this thesis had small sample sizes (although in relation to other studies using repeated measures analyses they are quite comparable) for each group. The patient samples were also ones of convenience and are not representative of all young, elderly, AD or PD populations. Although repeated measures analysis and

the MANOVA approach minimizes the sample size problem somewhat, and many such studies have much smaller sample sizes than those used in this thesis, the results should be generalized to the relevant populations only with caution. This is particularly true of the sample size in the AD(B) group (i.e., n = 4). It should also be noted that the subdivision of the AD sample [AD(A) vs AD(B)] was post-hoc and resulted from the findings of the study as it progressed. Nevertheless, the results of this small subsample provided informative data regarding the possible effects of progression of the disease on shifts of attention and motor programming. However, due to the difficulty the subjects [AD(B)] had in performing the experiments, it was not feasible, and did not seem appropriate, to try to gather more subjects with this level of cognitive impairment in order to increase the power of any statistical analyses.

Future Research

This thesis examined a number of issues and integrated three experimental perspectives (i.e., attention, motor functioning, and kinematics). As this has not been done previously, the current research may be considered to be exploratory in nature. The results have raised a number of issues that may be examined in future research. For example, the effects in this study may be replicated using larger samples, or using a larger number of trials (possibly eliminating the neutral trials) in order to increase the power

of the design in investigating differences in the "costs plus benefits". One might include a paradigm where exogenous cues are paired with exogenous targets, to examine whether there were even less differences among the groups in such a paradigm (compared with the Exogenous Paradigm used in this thesis). Various SOA's might be incorporated into the paradigm to examine the ability to maintain motor programs and/or shifts of attention, or to examine at what point, if any, differences among the groups become evident. Using PD subjects that are divided on the basis of disease severity (i.e., Hoehn and Yahr scores) and AD subjects based on level of cognitive impairment (e.g., MMSE scores) might provide further and better information (as in this study the AD groups were divided on their ability to do a task rather than on disease severity) about the effects of disease progression on shifts of visual attention and motor programming.

Future studies would benefit from conducting an analysis of the number and types of errors (e.g., anticipatory movements at the time of the cue, reaching to the wrong target on the invalid trials) made by the subjects. As well, an analysis of variable and constant error around the target would provide more detailed information about the end point accuracy of movements.

Future studies using a longitudinal approach where subjects were examined on these tasks a number of times over a specified period of time (e.g., each 3 months for one year)

would provide information on performance and disease progression.

In conclusion, this thesis has provided information about the effects of covert shifts of visual attention and the effects of informational precues on manual aiming movements in individuals who are, presumably, at their optimal level of physical abilities (i.e., young healthy adults), in individuals who have undergone the process of healthy aging, and in individuals who are undergoing the processes of the neurodegenerative disorders of AD and PD. Results of this thesis have also provided some (preliminary) indications as to the possible effects of the progression of AD on the processes of attention and motor function.

The most prominent finding in this thesis, in the authors opinion, is the importance of cognitive processes in motor function. In particular, that deficits in cognitive functioning (i.e., AD) can produce greater deficits in the production and execution of movements than motor deficits (PD). This finding has particularly important implications for physical rehabilitation programs.

Further, results of this thesis have provided an indication that the integration of the attention, motor function, and kinematic perspectives has merit for future examination of visual-motor processes and integration within and between numerous subject groups.

Appendix Ia

Instructions for Exogenous Target Paradigm

A condition is set up on the computer screen to demonstrate with as you give verbal instructions.

Here you see the numbers 1 to 4 on the top half of the computer screen (point to the numbers as you say this). You also see a dot in the centre of the screen (point to the dot).

We want you to start with your right index finger resting here (demonstrate) and then reach out and touch one of these numbers with your right index finger quickly and accurately (demonstrate).

Most of the time, we are going to give you a hint about which of these numbers we want you to point to. These hints will be arrows that point towards one of the numbers (demonstrate). Sometimes, you won't get a hint and a "plus" sign will show up instead.

The arrow or the plus sign will stay on for a short time and will then go out. The number that we want you to point to will then be surrounded by a circle. Even when you get a hint, it is very important that you look at the dot in the centre and keep looking at it until the circle comes on. That's because sometimes the circle will come on around a different number than the one the arrow pointed to. That won't happen very often, but it will happen sometimes. Remember, we always want you to point to the number in the circle.

What we want to know is how the hints can help you to do the task better.

We will practice this a few times so you can get used to the task. Have you any questions?

Appendix Ib

Instructions for Endogenous Target Paradigm

A condition is set up on the computer screen to demonstrate with as you give verbal instructions.

Here you see the numbers 1 to 4 on the top half of the computer screen (point to the numbers as you say this). You also see a dot in the centre of the screen (point to the dot).

We want you to start with your right index finger resting here (demonstrate) and then reach out and touch one of these numbers with your right index finger quickly and accurately (demonstrate).

Most of the time, we are going to give you a hint about which of these numbers we want you to point to. These hints will be arrows that point towards one of the numbers (demonstrate). Sometimes, you won't get a hint and a "plus" sign will show up instead.

The arrow or the "plus" sign will stay on for a short time and will then go out. The number that we want you to point to will then be shown to you at this dot. Even when you get a hint, it is very important that you look at the dot in the centre and keep looking at it until the number comes on. That's because sometimes the number that is shown at the dot will be a different number than the one the arrow pointed to. That won't happen very often, but it will happen sometimes. Remember, we always want you to point to the same number as the one that comes on at the dot.

What we want to know is how the hints can help you to do the task better.

We will practice this a few times so you can get used to the task. Have you any questions?

Appendix IIa

Table of least squares means and standard errors of the least squares means of the values presented in the figures for Study 1.

Appendix IIa

Figure #	Target Type	Measure	Condition	Mean	Standard Error
4.1	Exogenous	Onset (ms)	Valid	356.9	3.5
			Neutral	432.6	6.0
			Invalid	412.3	6.0
	Endogenous	Onset (ms)	Valid	416.8	3.5
			Neutral	521.6	6.0
			Invalid	491.0	6.0
4.2	Exogenous	Duration (ms)	Valid	577.8	2.2
			Neutral	583.8	3.7
			Invalid	590.0	3.9
	Endogenous	Duration (ms)	Valid	574.8	2.2
			Neutral	590.8	3.9
			Invalid	604.5	3.9
4.3	Exogenous	Peak Velocity (cm/sec)	Valid	90.6	0.3
			Neutral	90.2	0.5
			Invalid	89.3	0.5
	Endogenous	Peak Velocity (cm/sec)	Valid	91.6	0.3
			Neutral	89.9	0.5
			Invalid	88.6	0.5
4.4	Exogenous	Percent Deceleration	Valid	60.9	0.3
			Neutral	61.4	0.3
			Invalid	61.1	0.3
	Endogenous	Percent Deceleration	Valid	61.3	0.2
			Neutral	62.5	0.3
			Invalid	62.2	0.3
4.5	Exogenous	Resultant Error (mm)	Valid	7.9	0.1
			Neutral	8.0	0.2
			Invalid	7.8	0.2
	Endogenous	Resultant Error (mm)	Valid	7.5	0.1
			Neutral	7.4	0.1
			Invalid	7.7	0.2

Appendix IIb

Table of least squares means and standard errors of the least squares means of the values presented in the figures for Study 2.

Appendix IIb

Figure #	Group	Target Type	Measure	Condition	Mean	Standard Error
5.1	Elderly	Exogenous	Onset (ms)	Valid	512.3	6.9
				Neutral	632.7	11.8
				Invalid	613.8	11.9
	Elderly	Endogenous	Onset (ms)	Valid	612.3	6.9
				Neutral	701.9	11.9
				Invalid	675.7	11.9
5.2	Elderly	Exogenous	Duration (ms)	Valid	719.9	2.5
				Neutral	728.6	4.3
				Invalid	723.7	4.3
	Elderly	Endogenous	Duration (ms)	Valid	737.6	2.5
				Neutral	748.4	4.3
				Invalid	746.8	4.3
5.3	Elderly	Exogenous	Peak Velocity (cm/sec)	Valid	74.5	0.2
				Neutral	73.9	0.3
				Invalid	75.2	0.3
	Elderly	Endogenous	Peak Velocity (cm/sec)	Valid	72.5	0.2
				Neutral	71.7	0.3
				Invalid	71.9	0.3

Appendix IIb (Continued)

Figure #	Group	Target Type	Measure	Condition	Mean	Standard Error
5.4	Elderly	Exogenous	Percent Deceleration	Valid	59.4	0.2
				Neutral	59.9	0.4
				Invalid	60.4	0.4
	Elderly	Endogenous	Percent Deceleration	Valid	59.1	0.2
				Neutral	59.1	0.4
				Invalid	59.4	0.4
5.5	Elderly	Exogenous	Resultant Error (mm)	Valid	8.5	0.2
				Neutral	8.3	0.3
				Invalid	8.1	0.3
	Elderly	Endogenous	Resultant Error (mm)	Valid	8.4	0.2
				Neutral	8.5	0.3
				Invalid	8.4	0.3
5.6	Young	Exogenous	Onset (ms)	Valid	356.9	4.7
				Neutral	432.6	8.1
				Invalid	412.3	8.1
	Elderly	Exogenous	Onset (ms)	Valid	512.3	5.7
				Neutral	632.7	9.8
				Invalid	613.8	9.9

Appendix IIb (Continued)

Figure #	Group	Target Type	Measure	Condition	Mean	Standard Error
5.6	Young	Endogenous	Onset (ms)	Valid	416.8	4.7
				Neutral	521.6	8.1
				Invalid	491.0	8.1
	Elderly	Endogenous	Onset (ms)	Valid	612.3	5.7
				Neutral	701.9	9.9
				Invalid	675.7	9.8
5.7	Young	Exogenous	Duration (ms)	Valid	577.8	2.2
				Neutral	583.8	3.7
				Invalid	590.0	3.9
	Elderly	Exogenous	Duration (ms)	Valid	719.9	2.6
				Neutral	728.6	4.5
				Invalid	723.7	4.5
	Young	Endogenous	Duration (ms)	Valid	574.8	2.2
				Neutral	590.8	3.7
				Invalid	604.5	3.7
	Elderly	Endogenous	Duration (ms)	Valid	737.6	2.6
				Neutral	748.4	4.5
				Invalid	746.8	4.5

Appendix IIb (Continued)

Figure #	Group	Target Type	Measure	Condition	Mean	Standard Error
5.8	Young	Exogenous	Peak Velocity (cm/sec)	Valid	90.6	0.2
				Neutral	90.2	0.4
				Invalid	89.3	0.4
	Elderly	Exogenous	Peak Velocity (cm/sec)	Valid	74.5	0.3
				Neutral	73.9	0.5
				Invalid	75.2	0.2
	Young	Endogenous	Peak Velocity (cm/sec)	Valid	91.6	0.2
				Neutral	89.9	0.4
				Invalid	88.6	0.4
	Elderly	Endogenous	Peak Velocity (cm/sec)	Valid	72.5	0.3
				Neutral	71.7	0.5
				Invalid	71.9	0.5
5.9	Young	Exogenous	Percent Deceleration	Valid	60.9	0.2
				Neutral	61.4	0.3
				Invalid	61.1	0.3
	Elderly	Exogenous	Percent Deceleration	Valid	59.4	0.2
				Neutral	59.9	0.4
				Invalid	60.4	0.4

Appendix IIb (Continued)

Figure #	Group	Target Type	Measure	Condition	Mean	Standard Error
5.9	Young	Endogenous	Percent Deceleration	Valid	61.3	0.2
				Neutral	62.5	0.3
				Invalid	62.2	0.3
	Elderly	Endogenous	Percent Deceleration	Valid	59.1	0.2
				Neutral	59.1	0.4
				Invalid	59.4	0.4
5.10	Young	Exogenous	Resultant Error (mm)	Valid	7.9	0.1
				Neutral	8.0	0.2
				Invalid	7.8	0.2
	Elderly	Exogenous	Resultant Error (mm)	Valid	8.5	0.1
				Neutral	8.3	0.2
				Invalid	8.1	0.2
	Young	Endogenous	Resultant Error (mm)	Valid	7.5	0.1
				Neutral	7.4	0.2
				Invalid	7.7	0.2
	Elderly	Endogenous	Resultant Error (mm)	Valid	8.4	0.1
				Neutral	8.5	0.2
				Invalid	8.4	0.2

Appendix IIc

Table of least squares means and standard errors of the least squares means of the values presented in the figures for Study 3.

Appendix IIc

Figure #	Group	Target Type	Measure	Condition	Mean	Standard Error
6.1	AD(A)	Exogenous	Onset (ms)	Valid	655.2	24.8
				Neutral	907.4	41.1
				Invalid	881.3	40.6
	AD(A)	Endogenous	Onset (ms)	Valid	972.1	24.0
				Neutral	1277.4	40.9
				Invalid	1197.3	40.7
6.2	AD(A)	Exogenous	Duration (ms)	Valid	763.4	15.9
				Neutral	773.6	26.5
				Invalid	783.3	26.1
	AD(A)	Endogenous	Duration (ms)	Valid	789.2	15.5
				Neutral	817.5	26.4
				Invalid	910.6	26.2
6.3	AD(A)	Exogenous	Peak Velocity (cm/sec)	Valid	67.6	0.4
				Neutral	67.5	0.6
				Invalid	67.5	0.6
	AD(A)	Endogenous	Peak Velocity (cm/sec)	Valid	66.2	0.4
				Neutral	63.9	0.6
				Invalid	64.2	0.6

Appendix IIc (Continued)

Figure #	Group	Target Type	Measure	Condition	Mean	Standard Error
6.4	AD(A)	Exogenous	Percent Deceleration	Valid	60.7	0.4
				Neutral	61.7	0.7
				Invalid	61.0	0.7
	AD(A)	Endogenous	Percent Deceleration	Valid	61.1	0.3
				Neutral	61.3	0.6
				Invalid	61.7	0.6
6.5	AD(A)	Exogenous	Resultant Error (ms)	Valid	8.2	0.2
				Neutral	8.0	0.3
				Invalid	8.1	0.3
	AD(A)	Endogenous	Resultant Error (mm)	Valid	7.8	0.2
				Neutral	7.7	0.3
				Invalid	7.7	0.3
6.6	Elderly	Exogenous	Onset (ms)	Valid	512.3	12.9
				Neutral	632.7	22.2
				Invalid	613.8	22.4
	AD(A)	Exogenous	Onset (ms)	Valid	655.2	14.0
				Neutral	907.4	28.3
				Invalid	881.3	27.9

Appendix IIc (Continued)

Figure #	Group	Target Type	Measure	Condition	Mean	Standard Error
6.6	Elderly	Endogenous	Onset (ms)	Valid	612.3	12.9
				Neutral	701.9	22.3
				Invalid	675.7	22.2
	AD(A)	Endogenous	Onset (ms)	Valid	972.1	16.5
				Neutral	1277.4	28.2
				Invalid	1197.3	27.9
6.7	Elderly	Exogenous	Duration (ms)	Valid	719.9	7.8
				Neutral	728.6	13.4
				Invalid	723.7	13.5
	AD(A)	Exogenous	Duration (ms)	Valid	763.4	10.3
				Neutral	773.6	17.1
				Invalid	783.3	16.9
	Elderly	Endogenous	Duration (ms)	Valid	737.6	7.8
				Neutral	748.4	13.5
				Invalid	746.8	13.5
	AD(A)	Endogenous	Duration (ms)	Valid	789.2	9.9
				Neutral	817.5	17.0
				Invalid	910.6	16.9

Appendix IIc (Continued)

Figure #	Group	Target Type	Measure	Condition	Mean	Standard Error
6.8	Elderly	Exogenous	Peak Velocity (cm/sec)	Valid	74.5	0.3
				Neutral	73.9	0.4
				Invalid	75.2	0.4
	AD(A)	Exogenous	Peak Velocity (cm/sec)	Valid	67.6	0.3
				Neutral	67.5	0.5
				Invalid	67.5	0.5
	Elderly	Endogenous	Peak Velocity (cm/sec)	Valid	72.5	0.3
				Neutral	71.7	0.5
				Invalid	71.9	0.5
	AD(A)	Endogenous	Peak Velocity (cm/sec)	Valid	66.2	0.3
				Neutral	63.9	0.5
				Invalid	64.2	0.5
6.9	Elderly	Exogenous	Percent Deceleration	Valid	59.4	0.3
				Neutral	59.9	0.4
				Invalid	60.4	0.4
	AD(A)	Exogenous	Percent Deceleration	Valid	60.7	0.3
				Neutral	61.7	0.6
				Invalid	61.0	0.6

Appendix IIc (Continued)

Figure #	Group	Target Type	Measure	Condition	Mean	Standard Error
6.9	Elderly	Endogenous	Percent Deceleration	Valid	59.1	0.3
				Neutral	59.1	0.6
				Invalid	59.4	0.4
	AD(A)	Endogenous	Percent Deceleration	Valid	61.1	0.3
				Neutral	61.3	0.6
				Invalid	61.7	0.6
6.10	Elderly	Exogenous	Resultant Error (mm)	Valid	8.5	0.1
				Neutral	8.3	0.2
				Invalid	8.1	0.2
	AD(A)	Exogenous	Resultant Error (ms)	Valid	8.2	0.2
				Neutral	8.0	0.3
				Invalid	8.1	0.3
	Elderly	Endogenous	Resultant Error (mm)	Valid	8.4	0.1
				Neutral	8.5	0.2
				Invalid	8.4	0.2
	AD(A)	Endogenous	Resultant Error (mm)	Valid	7.8	0.2
				Neutral	7.7	0.3
				Invalid	7.7	0.3

Appendix IIc (Continued)

Figure #	Group	Target Type	Measure	Condition	Mean	Standard Error
6.11	AD(A)	Exogenous	Onset (ms)	Valid	655.2	33.2
				Neutral	907.4	55.2
				Invalid	881.3	54.4
	AD(B)	Exogenous	Onset (ms)	Valid	1279.8	66.0
				Neutral	1599.6	110.4
				Invalid	1578.6	113.2
6.12	AD(A)	Exogenous	Duration (ms)	Valid	763.4	6.5
				Neutral	773.6	10.8
				Invalid	783.3	10.7
	AD(B)	Exogenous	Duration (ms)	Valid	859.9	12.8
				Neutral	900.9	21.4
				Invalid	981.3	21.9
6.13	AD(A)	Exogenous	Peak Velocity (cm/sec)	Valid	65.6	0.4
				Neutral	67.5	0.6
				Invalid	67.5	0.6
	AD(B)	Exogenous	Peak Velocity (cm/sec)	Valid	54.8	0.8
				Neutral	55.3	1.3
				Invalid	53.7	1.3

Appendix IIc (Continued)

Figure #	Group	Target Type	Measure	Condition	Mean	Standard Error
6.14	AD(A)	Exogenous	Percent Deceleration	Valid	60.7	0.5
				Neutral	61.7	0.8
				Invalid	61.0	0.8
	AD(B)	Exogenous	Percent Deceleration	Valid	63.6	0.9
				Neutral	65.8	1.6
				Invalid	64.0	1.6
6.15	AD(A)	Exogenous	Resultant Error (mm)	Valid	8.2	0.1
				Neutral	8.0	0.2
				Invalid	8.2	0.2
	AD(B)	Exogenous	Resultant Error (mm)	Valid	7.4	0.2
				Neutral	7.8	0.3
				Invalid	8.4	0.4

Appendix IId

Table of least squares means and standard errors of the least squares means of the values presented in the figures for Study 4.

Appendix IId

Figure #	Group	Target Type	Measure	Condition	Mean	Standard Error
7.1	PD	Exogenous	Onset (ms)	Valid	579.9	14.8
				Neutral	709.8	25.3
				Invalid	668.0	25.3
	PD	Endogenous	Onset (ms)	Valid	796.5	14.8
				Neutral	966.3	25.5
				Invalid	821.1	25.3
7.2	PD	Exogenous	Duration (ms)	Valid	754.9	4.9
				Neutral	796.3	8.4
				Invalid	782.7	8.4
	PD	Endogenous	Duration (ms)	Valid	778.3	4.9
				Neutral	815.9	8.5
				Invalid	826.3	8.4
7.3	PD	Exogenous	Peak Velocity (cm/sec)	Valid	65.8	0.3
				Neutral	65.8	0.5
				Invalid	66.0	0.5
	PD	Endogenous	Peak Velocity (cm/sec)	Valid	63.0	0.3
				Neutral	61.3	0.5
				Invalid	62.6	0.5

Appendix IId (Continued)

Figure #	Group	Target Type	Measure	Condition	Mean	Standard Error
7.4	PD	Exogenous	Percent Deceleration	Valid	59.4	0.3
				Neutral	59.1	0.5
				Invalid	60.2	0.5
	PD	Endogenous	Percent Deceleration	Valid	58.1	0.3
				Neutral	57.7	0.5
				Invalid	58.2	0.5
7.5	PD	Exogenous	Resultant Error (mm)	Valid	10.1	0.2
				Neutral	9.8	0.3
				Invalid	9.6	0.3
	PD	Endogenous	Resultant Error (mm)	Valid	10.4	0.2
				Neutral	9.6	0.4
				Invalid	13.7	0.3
7.6	Elderly	Exogenous	Onset (ms)	Valid	512.3	12.8
				Neutral	632.7	22.2
				Invalid	613.8	22.2
	AD(A)	Exogenous	Onset (ms)	Valid	655.2	16.9
				Neutral	907.4	28.1
				Invalid	881.3	27.7
	PD	Exogenous	Onset (ms)	Valid	579.9	15.0
				Neutral	709.8	25.8
				Invalid	668.0	25.7

Appendix IId (Continued)

Figure #	Group	Target Type	Measure	Condition	Mean	Standard Error
7.6	Elderly	Endogenous	Onset (ms)	Valid	612.3	12.8
				Neutral	701.9	22.1
				Invalid	675.7	22.1
	AD(A)	Endogenous	Onset (ms)	Valid	972.1	16.4
				Neutral	1277.4	27.9
				Invalid	1197.3	27.8
	PD	Endogenous	Onset (ms)	Valid	796.5	15.1
				Neutral	966.3	26.0
				Invalid	821.1	25.8
7.7	Elderly	Exogenous	Duration (ms)	Valid	719.9	6.9
				Neutral	728.6	11.9
				Invalid	723.7	11.9
	AD(A)	Exogenous	Duration (ms)	Valid	763.4	9.1
				Neutral	773.6	15.1
				Invalid	783.3	14.9
	PD	Exogenous	Duration (ms)	Valid	754.9	8.1
				Neutral	796.3	13.9
				Invalid	782.7	13.9

Appendix IId (Continued)

Figure #	Group	Target Type	Measure	Condition	Mean	Standard Error
7.7	Elderly	Endogenous	Duration (ms)	Valid	737.6	6.9
				Neutral	748.4	11.9
				Invalid	746.8	11.9
	AD(A)	Endogenous	Duration (ms)	Valid	789.2	8.4
				Neutral	817.5	15.1
				Invalid	910.6	14.9
	PD	Endogenous	Duration (ms)	Valid	778.3	8.2
				Neutral	815.9	14.0
				Invalid	826.3	13.9
7.8	Elderly	Exogenous	Peak Velocity (cm/sec)	Valid	74.5	0.3
				Neutral	73.9	0.4
				Invalid	75.2	0.4
	AD(A)	Exogenous	Peak Velocity (cm/sec)	Valid	67.6	0.3
				Neutral	67.5	0.5
				Invalid	67.5	0.5
	PD	Exogenous	Peak Velocity (cm/sec)	Valid	65.8	0.3
				Neutral	65.8	0.5
				Invalid	66.0	0.5

Appendix IId (Continued)

Figure #	Group	Target Type	Measure	Condition	Mean	Standard Error
7.8	Elderly	Endogenous	Peak Velocity (cm/sec)	Valid	72.5	0.2
				Neutral	71.7	0.4
				Invalid	71.9	0.4
	AD(A)	Endogenous	Peak Velocity (cm/sec)	Valid	66.2	0.3
				Neutral	63.9	0.5
				Invalid	64.2	0.5
	PD	Endogenous	Peak Velocity (cm/sec)	Valid	63.0	0.3
				Neutral	61.3	0.5
				Invalid	62.6	0.5
7.9	Elderly	Exogenous	Percent Deceleration	Valid	59.4	0.3
				Neutral	59.9	0.4
				Invalid	60.4	0.4
	AD(A)	Exogenous	Percent Deceleration	Valid	60.7	0.3
				Neutral	61.7	0.6
				Invalid	61.0	0.6
	PD	Exogenous	Percent Deceleration	Valid	59.4	0.3
				Neutral	59.1	0.5
				Invalid	60.2	0.5

Appendix IId (Continued)

Figure #	Group	Target Type	Measure	Condition	Mean	Standard Error
7.9	Elderly	Endogenous	Percent Deceleration	Valid	59.1	0.3
				Neutral	59.1	0.4
				Invalid	60.4	0.4
	AD(A)	Endogenous	Percent Deceleration	Valid	61.1	0.3
				Neutral	61.3	0.6
				Invalid	61.7	0.6
	PD	Endogenous	Percent Deceleration	Valid	58.1	0.3
				Neutral	57.7	0.5
				Invalid	58.2	0.5
7.10	Elderly	Exogenous	Resultant Error (mm)	Valid	8.5	0.1
				Neutral	8.3	0.3
				Invalid	8.1	0.3
	AD(A)	Exogenous	Resultant Error (mm)	Valid	8.2	0.2
				Neutral	8.0	0.3
				Invalid	8.1	0.3
	PD	Exogenous	Resultant Error (mm)	Valid	10.1	0.2
				Neutral	9.8	0.3
				Invalid	9.6	0.3

Appendix IId (Continued)

Figure #	Group	Target Type	Measure	Condition	Mean	Standard Error
7.10	Elderly	Endogenous	Resultant Error (mm)	Valid	8.4	0.1
				Neutral	8.5	0.3
				Invalid	8.4	0.3
	AD(A)	Endogenous	Resultant Error (mm)	Valid	7.8	0.2
				Neutral	7.7	0.3
				Invalid	7.7	0.3
	PD	Endogenous	Resultant Error (mm)	Valid	10.4	0.2
				Neutral	9.6	0.4
				Invalid	13.7	0.3

Appendix IIIa

Table of F ratios and Probability levels for mixed model analysis of variance based on results of multivariate analysis of variance (SAS, GLM program) in Study 1. Probability levels are based on 2, 28 degrees of freedom for condition (C), 1, 14 degrees of freedom for target type (T), and 2, 28 degrees of freedom for condition by target type (C by T). Probability levels less than .05 are indicated by "*". Dependent measures are Movement Onset, Movement Duration, Peak Velocity, Percent Deceleration, and Resultant Error.

Appendix IIIa

	Onset		Movement Duration		Peak Velocity	
Source	F ratio	Probability	F ratio	Probability	F ratio	Probability
C	35.2	0.0001*	7.92	0.0019*	11.7	0.0002*
T	52.1	0.0001*	.194	0.6666	.001	0.9830
C by T	5.15	0.0125*	4.26	0.0243*	3.08	0.0620

	Percent Deceleration		Resultant Error	
Source	F ratio	Probability	F ratio	Probability
C	3.29	0.0520	.166	0.8481
T	1.79	0.2018	2.91	0.1104
C by T	2.03	0.1503	.796	0.4611

Appendix IIIb

Table of F ratios and Probability levels for Mixed Model Analysis of Variance based on results of Multivariate Analysis of Variance (SAS, GLM program) in Study 2 (elderly vs young). Probability levels are based on 1, 28 degrees of freedom for Group (G), Target type (T), and Group by Target Type (G by T), and 2, 56 degrees of freedom for Condition (C) and Group by Condition (G by C). Probability levels less than .05 are indicated by "*". Dependent measures are Movement Onset, Movement Duration, Peak Velocity, Percent Deceleration, and Resultant Error.

Appendix IIIb

Source	Onset		Movement Duration		Peak Velocity	
	F ratio	Probability	F ratio	Probability	F ratio	Probability
G	28.7	0.0001*	18.2	0.0002*	10.9	0.0027*
C	52.7	0.0001*	9.11	0.0004*	7.39	0.0014*
G by C	.463	0.6315	2.05	0.1375	5.80	0.0050*
T	53.6	0.0001*	1.43	0.2419	1.11	0.3017
G by T	.003	0.9536	.407	0.5288	1.04	0.3168
C by T	.449	0.6400	3.16	0.0495*	4.51	0.0149*
G by C by T	6.48	0.0029*	.953	0.3914	.611	0.5459

Continued...

Appendix IIIb (Continued)

	Percent Deceleration		Resultant Error	
Source	F ratio	Probability	F ratio	Probability
G	2.79	0.1061	.584	0.4511
C	4.24	0.0190*	.055	0.9465
G by C	.810	0.4499	.492	0.6138
T	.031	0.8620	.217	0.6450
G by T	2.59	0.1185	1.19	0.2848
C by T	.014	0.9862	.862	0.4276
G by C by T	3.01	0.0570	.377	0.6873

Appendix IIIc

Table of F ratios and Probability levels for Mixed Model Analysis of Variance based on results of Multivariate Analysis of Variance (SAS, GLM program) in Study 3 for the Elderly control and the AD(A) subject group. Probability levels are based on 1, 23 degrees of freedom for Group (G), Target Type (T), and Group by Target Type (G by T), and 2, 46 degrees of freedom for Condition (C) and Group by Condition (G by C), and for Group by Condition by Target Type (G by C by T). Probability levels less than .05 are indicated by "*". Dependent measures are Movement Onset, Movement Duration, Peak Velocity, Percent Deceleration, and Resultant Error.

Appendix IIIc

	Onset		Movement Duration		Peak Velocity	
Source	F ratio	Probability	F ratio	Probability	F ratio	Probability
G	10.2	0.0041*	4.80	0.0388*	4.11	0.0544
C	45.5	0.0001*	9.92	0.0003*	3.31	0.0453*
G by C	9.58	0.0003*	6.99	0.0022*	1.04	0.3623
T	26.3	0.0001*	5.74	0.0250*	4.62	0.0423*
G by T	10.4	0.0037	1.63	0.2142	.007	0.9338
C by T	.387	0.6810	4.56	0.0155*	5.22	0.0088*
G by C by T	1.07	0.3517	3.78	0.0302*	1.71	0.1919

Continued...

Appendix IIIc (Continued)

	Percent Deceleration		Resultant Error	
Source	F ratio	Probability	F ratio	Probability
G	1.84	0.1882	.264	0.6126
C	1.97	0.1512	.283	0.7545
G by C	.231	0.7943	.159	0.8536
T	.163	0.6906	.067	0.7988
G by T	.696	0.4126	.377	0.5452
C by T	.634	0.5300	.317	0.7297
G by C by T	.495	0.6127	.268	0.7661

Appendix IIId

Table of F ratios and Probability levels for Mixed Model Analysis of Variance based on results of Multivariate Analysis of Variance (SAS, GLM program) in Study 3 for the AD(A) and the AD(B) subject groups. Probability levels are based on 1, 12 degrees of freedom for Group (G), and 2, 30 degrees of freedom for Condition (C) and Group by Condition (G by C). Probability levels less than .05 are indicated by "*". Dependent measures are Movement Onset, Movement Duration, Peak Velocity, Percent Deceleration, and Resultant Error.

Appendix IIId

	Onset		Movement Duration		Peak Velocity	
Source	F ratio	Probability	F ratio	Probability	F ratio	Probability
G	13.8	0.0029*	8.12	0.0143*	5.96	0.0309*
C	14.6	0.0001*	13.4	0.0001*	.373	0.6913
G by C	.227	0.7981	7.02	0.0025*	.350	0.7071

	Percent Deceleration		Resultant Error	
Source	F ratio	Probability	F ratio	Probability
G	2.49	0.1395	.023	0.8810
C	1.33	0.2777	1.50	0.2305
G by C	.220	0.8038	1.79	0.1730

Appendix IIIe

Table of F ratios and Probability levels for Mixed Model Analysis of Variance based on results of Multivariate Analysis of Variance (SAS, GLM program) in Study 4 (elderly, AD, PD). Probability levels are based on 2, 33 degrees of freedom for Group (G), 1, 33 degrees of freedom for Target Type (T), 2, 33 degrees of freedom for Group by Target Type (G by T), 2, 66 degrees of freedom for Condition (C), and 4, 66 degrees of freedom for Group by Condition (C by C) and for Group by Condition by Target Type (G by C by T). Probability levels less than .05 are indicated by "*". Dependent measures are Movement Onset, Movement Duration, Peak Velocity, Percent Deceleration, and Resultant Error.

Appendix IIIe

	Onset		Movement Duration		Peak Velocity	
Source	F ratio	Probability	F ratio	Probability	F ratio	Probability
G	4.32	0.0215*	2.50	0.0973	2.89	0.0697
C	53.8	0.0001*	17.2	0.0003*	4.32	0.0172*
G by C	5.94	0.0004*	4.53	0.0027*	.544	0.7037
T	28.8	0.0001*	8.06	0.0077*	11.21	0.0020*
G by T	3.89	0.0304*	1.01	0.3718	.136	0.8732
C by T	1.83	0.1681	5.44	0.0065*	6.77	0.0021*
G by C by T	.937	0.4480	2.56	0.0464*	1.37	0.2537

Continued...

Appendix IIIe (Continued)

	Percent Deceleration		Resultant Error	
Source	F ratio	Probability	F ratio	Probability
G	1.33	0.2770	2.01	0.1496
C	1.49	0.2324	1.10	0.3393
G by C	.425	0.7904	.732	0.5732
T	2.20	0.1477	.027	0.8695
G by T	1.19	0.3167	.326	0.7243
C by T	.698	0.5011	.834	0.4384
G by C by T	.390	0.8153	.898	0.4701

Appendix IIIf

Table of F ratios and Probability levels for Mixed Model Analysis of Variance based on results of Multivariate Analysis of Variance (SAS, GLM program) in Study 1 of Laterality Effects (based on observations from the valid, invalid, and neutral cue conditions). Probability levels are based on 1, 14 degrees of freedom for laterality (L). Probability levels less than .05 are indicated by "*". Dependent measures are Movement Onset (IRED #1), Movement Onset (IRED #2), Movement Duration, Peak Velocity, Percent Deceleration, and Resultant Error.

Appendix IIIf

	Onset (IRED#1)		Onset (IRED #2)		Movement Duration	
Source	F ratio	Probability	F ratio	Probability	F ratio	Probability
L	.258	0.6196	7.49	0.0160*	73.8	0.0001*

	Peak Velocity		Percent Deceleration		Resultant Error	
Source	F ratio	Probability	F ratio	Probability	F ratio	Probability
L	50.3	0.0001*	8.81	0.0102*	4.38	0.0551

Appendix IIIg

Table of F ratios and Probability levels for Mixed Model Analysis of Variance based on results of Multivariate Analysis of Variance (SAS, GLM program) in Study 1 of Laterality Effects (based on observations from the neutral cue condition only). Probability levels are based on 1, 14 degrees of freedom for Laterality (L). Probability levels less than .05 are indicated by "*". Dependent measures are Movement Onset (IRED #1), Movement Duration, Peak Velocity, Percent Deceleration, and Resultant Error.

Appendix IIIg

	Onset (IRED #1)		Movement Duration		Peak Velocity	
Source	F ratio	Probability	F ratio	Probability	F ratio	Probability
L	6.82	0.0205*	44.8	0.0001*	48.7	0.0001*

	Percent Deceleration		Resultant Error	
Source	F ratio	Probability	F ratio	Probability
L	8.13	0.0128*	4.58	0.0505

Appendix IIIh

Table of F ratios and Probability levels for Mixed Model Analysis of Variance based on results of Multivariate Analysis of Variance (SAS, GLM program) in Study 2 of Laterality Effects. Probability levels are based on 1, 28 degrees of freedom for group (G), and 1, 29 degrees of freedom for laterality (L) and group by laterality (G by L). Probability levels less than .05 are indicated by "*". Dependent measures are Movement Onset, Movement Duration, Peak Velocity, Percent Deceleration, and Resultant Error.

Appendix IIIh

Source	Onset		Movement Duration		Peak Velocity	
	F ratio	Probability	F ratio	Probability	F ratio	Probability
G	28.4	0.0001*	18.1	0.0002*	11.0	0.0025*
L	10.4	0.0031*	143	0.0001*	99.5	0.0001*
G by L	1.66	0.2076	.435	0.5149	.264	0.6118

Source	Percent Deceleration		Resultant Error	
	F ratio	Probability	F ratio	Probability
G	2.50	0.1252	.662	0.4227
L	13.15	0.0011*	1.27	0.2686
G by L	2.04	0.1641	1.33	0.2587

Appendix IV (a to l)

Tables of F ratios and Probability levels for Mixed Model Analysis of Variance based on results of Multivariate Analysis of Variance (SAS, GLM program) in Study 1 and Study 2 of Meridian Effects. Probability levels are based on 1, 14 degrees of freedom for meridian (M). Probability levels less than .05 are indicated by "*". Dependent measures are Movement Onset, Movement Duration, Peak Velocity, Percent Deceleration, and Resultant Error. Appendix IVa is for the Exogenous Target Paradigm and contralateral targets for the young subject group. Appendix IVb is for the Endogenous Target Paradigm and contralateral targets for the young subject group. Appendix IVc is for the Exogenous Target Paradigm and ipsilateral targets for the young subject group. Appendix IVd is for the Endogenous Target Paradigm and ipsilateral targets for the young subject group. Appendix IVe is for the Exogenous Target Paradigm and contralateral targets for the elderly subject group. Appendix IVf is for the Endogenous Target Paradigm and contralateral targets for the elderly subject group. Appendix IVf is for the Exogenous Target Paradigm and ipsilateral targets for the elderly subject group. Appendix IVg is for the Endogenous Target Paradigm and ipsilateral targets for the elderly subject group. Appendix IVh-l are for the data for the young and elderly groups combined. In this analysis, probability levels are based on 1, 31 degrees of freedom.

Appendix IVa

Group 1 (Exogenous Paradigm - Contralateral Targets)

	Onset		Movement Duration		Peak Velocity	
Source	F ratio	Probability	F ratio	Probability	F ratio	Probability
M	0.8417	0.3743	0.0684	0.7975	0.6152	0.4458

	Percent Deceleration		Resultant Error	
Source	F ratio	Probability	F ratio	Probability
M	2.059	0.1731	0.2466	0.6272

Appendix IVb

Group 1 (Endogenous Paradigm - Contralateral Targets)

	Onset		Movement Duration		Peak Velocity	
Source	F ratio	Probability	F ratio	Probability	F ratio	Probability
M	0.4505	0.5129	0.0787	0.7832	0.0388	0.8466

	Percent Deceleration		Resultant Error	
Source	F ratio	Probability	F ratio	Probability
M	0.8880	0.3619	4.7074	0.0477*

Appendix IVc

Group 1 (Exogenous Paradigm - Ipsilateral Targets)

	Onset		Movement Duration		Peak Velocity	
Source	F ratio	Probability	F ratio	Probability	F ratio	Probability
M	0.0886	0.7703	5.7617	0.0308*	3.8615	0.0695

	Percent Deceleration		Resultant Error	
Source	F ratio	Probability	F ratio	Probability
M	0.1771	0.6802	0.0004	0.9833

Appendix IVd

Group 1 (Endogenous Paradigm - Ipsilateral Targets)

	Onset		Movement Duration		Peak Velocity	
Source	F ratio	Probability	F ratio	Probability	F ratio	Probability
M	0.0257	0.8750	2.0910	0.8410	0.6459	0.4347

	Percent Deceleration		Resultant Error	
Source	F ratio	Probability	F ratio	Probability
M	0.0806	0.7805	0.0348	0.8547

Appendix IVe

Group 2 (Exogenous Paradigm - Contralateral Targets)

	Onset		Movement Duration		Peak Velocity	
Source	F ratio	Probability	F ratio	Probability	F ratio	Probability
M	0.0103	0.9205	0.5057	0.4882	0.2309	0.6377

	Percent Deceleration		Resultant Error	
Source	F ratio	Probability	F ratio	Probability
M	1.9912	0.1789	2.1029	0.1682

Appendix IVf

Group 2 (Endogenous Paradigm - Contralateral Targets)

Source	Onset		Movement Duration		Peak Velocity	
	F ratio	Probability	F ratio	Probability	F ratio	Probability
M	0.7544	0.3985	0.4788	0.5000	0.3982	0.5380

Source	Percent Deceleration		Resultant Error	
	F ratio	Probability	F ratio	Probability
M	1.5642	0.2304	0.2842	0.6018

Appendix IVg

Group 2 (Exogenous Paradigm - Ipsilateral Targets)

	Onset		Movement Duration		Peak Velocity	
Source	F ratio	Probability	F ratio	Probability	F ratio	Probability
M	1.8105	0.1995	0.4663	0.5056	0.4451	0.5154

	Percent Deceleration		Resultant Error	
Source	F ratio	Probability	F ratio	Probability
M	1.8562	0.1936	2.2347	0.1566

Appendix IVh

Group 2 (Endogenous Paradigm - Ipsilateral Targets)

	Onset		Movement Duration		Peak Velocity	
Source	F ratio	Probability	F ratio	Probability	F ratio	Probability
M	1.3467	0.2649	0.3994	0.5373	5.0040	0.0415*

	Percent Deceleration		Resultant Error	
Source	F ratio	Probability	F ratio	Probability
M	0.4888	0.4957	1.6065	0.2251

Appendix IVi

Group 2 & 3 (Exogenous Paradigm - Contralateral Targets)

Source	Onset		Movement Duration		Peak Velocity	
	F ratio	Probability	F ratio	Probability	F ratio	Probability
M	0.0362	0.8503	0.2438	0.6249	0.0370	0.8484

Source	Percent Deceleration		Resultant Error	
	F ratio	Probability	F ratio	Probability
M	0.0816	0.7769	0.2843	0.5975

Appendix IVj

Group 2 & 3 (Endogenous Paradigm - Contralateral Targets)

	Onset		Movement Duration		Peak Velocity	
Source	F ratio	Probability	F ratio	Probability	F ratio	Probability
M	1.2498	0.2709	0.2783	0.6015	0.0761	0.7845

	Percent Deceleration		Resultant Error	
Source	F ratio	Probability	F ratio	Probability
M	0.0000	0.9971	0.8296	0.3692

Appendix IVk

Group 2 & 3 (Exogenous Paradigm - Ipsilateral Targets)

	Onset		Movement Duration		Peak Velocity	
Source	F ratio	Probability	F ratio	Probability	F ratio	Probability
M	1.4821	0.2326	4.4105	0.0439*	3.2386	0.0816

	Percent Deceleration		Resultant Error	
Source	F ratio	Probability	F ratio	Probability
M	1.3849	0.2473	1.6184	0.2120

Appendix IV1

Group 2 & 3 (Endogenous Paradigm - Ipsilateral Targets)

	Onset		Movement Duration		Peak Velocity	
Source	F ratio	Probability	F ratio	Probability	F ratio	Probability
M	1.1992	0.2818	1.7465	0.1957	4.0180	0.0530

	Percent Deceleration		Resultant Error	
Source	F ratio	Probability	F ratio	Probability
M	0.5960	0.4456	1.0764	0.3073

Appendix V

Table of the means and standard deviations for each of the dependent variables for each cue/target combination (for each of the groups) used in this thesis. These means and standard deviations were derived by calculating the mean for each subject for each condition and then calculating the mean for all the subjects.

APPENDIX V

GROUP 1 (Young) - Exogenous Target Paradigm

		RT		MT		PV		% Dec		Error	
Cue	Target	M	SD	M	SD	M	SD	M	SD	M	SD
0	1	438.6	74.3	613.5	109.7	80.2	14.9	59.8	4.1	6.9	1.9
0	2	414.8	88.1	604.4	114.9	90.2	16.8	59.9	4.5	8.0	3.0
0	3	445.7	129.9	577.8	109.9	97.1	17.5	62.7	4.6	8.2	3.1
0	4	429.2	71.3	541.4	92.9	93.1	17.6	63.4	4.5	8.9	3.2
1	1	354.2	92.5	611.1	110.9	80.9	14.9	58.7	4.1	7.1	2.2
1	2	395.3	81.0	604.6	115.2	89.4	16.6	58.3	4.4	7.4	3.6
1	3	418.2	83.3	580.4	112.4	94.9	16.3	61.4	5.6	8.0	4.3
1	4	405.4	82.0	562.3	97.6	93.2	16.7	64.6	5.6	8.6	3.5
2	1	406.8	89.4	636.6	153.9	78.9	15.7	59.7	6.1	7.1	3.2
2	2	351.0	93.2	595.3	123.4	90.4	16.8	59.1	3.8	8.2	3.2
2	3	404.9	73.2	557.1	100.6	97.7	18.7	61.3	5.9	7.6	3.7
2	4	429.3	77.6	538.1	101.0	92.7	17.3	63.2	3.8	9.3	5.2

APPENDIX V

GROUP 1 (Young) (Con't)

Exogenous Target Paradigm

		RT		MT		PV		% Dec		Error	
Cue	Target	M	SD	M	SD	M	SD	M	SD	M	SD
3	1	409.4	67.4	624.6	111.8	79.5	14.7	59.6	5.1	6.5	1.8
3	2	409.2	73.9	607.8	126.9	88.5	16.8	59.7	5.4	7.9	4.3
3	3	364.7	92.3	567.6	113.1	97.2	17.4	62.2	4.3	7.5	2.6
3	4	416.4	123.5	545.4	98.5	91.7	19.2	61.9	6.7	8.1	4.6
4	1	419.7	57.4	618.5	117.5	91.3	14.6	60.6	4.1	7.4	2.6
4	2	429.9	93.3	608.9	113.6	89.6	17.9	60.3	4.8	8.7	5.3
4	3	401.0	77.5	594.3	121.8	94.9	17.6	61.7	5.6	7.6	3.9
4	4	357.9	81.4	537.4	96.5	94.1	18.1	63.6	4.8	8.9	3.6

APPENDIX V (Continued) - GROUP 1 (Young)

Endogenous Target Paradigm

		RT		MT		PV		% Dec		Error	
Cue	Target	M	SD	M	SD	M	SD	M	SD	M	SD
0	1	509.7	86.6	614.5	116.5	79.6	16.0	61.1	3.9	7.0	2.0
0	2	515.5	86.6	610.8	133.7	88.8	18.3	60.9	4.2	6.9	2.5
0	3	523.9	81.1	575.5	117.7	97.0	19.5	63.4	5.5	7.3	2.2
0	4	536.5	103.8	561.8	102.6	94.3	21.6	64.5	6.2	8.5	3.2
1	1	412.4	83.7	602.6	126.8	81.4	16.2	59.2	4.0	6.9	2.4
1	2	504.5	96.7	624.3	120.6	88.9	18.3	61.5	5.6	7.6	3.2
1	3	490.4	89.6	613.2	107.6	92.6	15.9	62.7	4.5	8.6	3.7
1	4	530.3	117.1	563.7	95.4	92.6	19.6	64.5	5.1	8.2	3.8
2	1	465.8	78.3	640.6	106.3	78.5	15.5	60.2	4.9	7.5	3.8
2	2	418.8	78.5	590.5	123.7	90.6	17.7	60.1	4.2	7.1	2.6

APPENDIX V (Continued) - GROUP 1 (Young)

Endogenous Target Paradigm

		RT		MT		PV		% Dec		Error	
Cue	Target	M	SD	M	SD	M	SD	M	SD	M	SD
2	3	475.2	108.2	603.1	107.0	92.5	16.8	62.1	6.0	8.0	2.5
2	4	498.4	87.3	575.6	107.8	92.6	22.1	63.8	5.3	8.2	3.3
3	1	481.2	83.5	633.5	101.2	79.9	16.1	60.9	5.4	7.4	3.8
3	2	514.1	86.4	620.3	125.7	89.4	17.3	63.0	5.4	6.0	1.8
3	3	423.9	80.1	567.4	118.5	98.6	21.0	62.7	5.8	7.7	2.1
3	4	484.7	84.6	559.4	102.2	93.3	18.4	62.8	8.1	8.0	3.6
4	1	474.8	56.7	610.2	120.5	80.4	16.6	61.1	5.1	7.6	2.7
4	2	494.7	55.3	618.7	134.7	88.9	18.7	60.8	5.5	7.0	2.8
4	3	477.8	92.3	535.3	110.3	93.9	17.1	62.4	6.5	8.2	4.2
4	4	411.8	75.7	537.9	101.8	95.8	20.2	63.3	6.3	8.0	2.8

APPENDIX V (Continued) - GROUP 2 (Elderly)

Exogenous Target Paradigm

		RT		MT		PV		% Dec		Error	
Cue	Target	M	SD	M	SD	M	SD	M	SD	M	SD
0	1	628.2	132.9	760.1	89.9	65.3	7.1	59.8	4.7	8.7	3.6
0	2	606.6	193.4	752.4	92.1	72.3	9.1	59.4	3.4	8.3	3.2
0	3	677.3	176.4	705.0	90.1	80.7	10.6	59.7	3.7	8.4	2.9
0	4	616.9	148.1	695.7	130.4	77.4	13.0	60.9	4.9	7.9	2.7
1	1	512.2	131.3	747.8	93.7	66.5	7.5	57.6	4.0	8.9	2.9
1	2	602.5	167.9	761.0	81.7	74.6	9.1	62.2	7.8	8.4	4.1
1	3	669.2	319.9	703.3	109.1	81.1	11.2	60.6	5.0	7.7	3.8
1	4	642.7	223.9	719.4	123.3	76.5	12.5	61.6	5.7	8.1	2.8
2	1	598.3	219.3	777.7	112.1	66.0	12.1	59.8	7.6	8.8	3.9
2	2	495.8	106.9	741.3	80.3	73.7	9.5	59.2	4.5	8.2	2.7
2	3	538.0	109.7	696.5	100.1	82.6	13.0	59.3	5.2	7.1	3.6

APPENDIX V (Continued) - GROUP 2 (Elderly)

Exogenous Target Paradigm

		RT		MT		PV		% Dec		Error	
Cue	Target	M	SD	M	SD	M	SD	M	SD	M	SD
2	4	631.2	283.5	680.3	133.6	77.9	14.2	61.9	3.8	7.3	4.1
3	1	608.3	218.9	739.3	76.9	69.9	8.6	58.6	4.5	8.7	5.4
3	2	597.5	182.2	746.8	107.5	75.2	11.4	60.3	5.1	7.2	3.3
3	3	525.5	148.1	708.1	96.2	80.4	11.3	59.8	3.2	8.2	2.8
3	4	612.5	212.5	677.7	98.2	78.9	12.7	60.8	5.2	8.4	3.7
4	1	668.4	298.5	722.8	91.2	67.2	9.3	57.9	4.9	8.6	3.9
4	2	627.8	211.9	752.2	109.3	73.3	9.4	60.8	6.1	8.6	3.2
4	3	586.7	189.2	707.9	82.3	81.6	11.6	60.7	4.8	8.3	4.3
4	4	516.1	116.1	682.4	98.6	77.6	12.4	60.9	3.6	8.4	2.5

APPENDIX V (Continued) - GROUP 2 (Elderly)

Endogenous Target Paradigm

		RT		MT		PV		% Dec		Error	
Cue	Target	M	SD	M	SD	M	SD	M	SD	M	SD
0	1	698.9	123.9	794.4	84.3	63.3	8.4	58.6	4.1	8.8	3.2
0	2	693.4	128.5	759.1	81.5	71.5	8.8	59.2	5.2	7.6	3.4
0	3	722.5	95.4	730.2	81.6	77.4	11.3	58.6	5.7	7.9	3.2
0	4	690.7	104.2	706.3	95.6	75.0	11.3	60.1	5.6	9.9	5.4
1	1	581.6	116.7	769.6	77.3	64.3	7.3	57.8	3.8	8.3	3.1
1	2	650.2	175.3	757.8	62.4	72.3	9.8	58.4	6.1	8.3	3.8
1	3	669.9	174.9	749.9	88.1	76.9	11.9	58.8	5.1	8.8	4.1
1	4	764.0	278.0	716.4	92.4	74.7	10.9	60.7	4.2	9.4	4.9
2	1	653.6	127.5	791.1	72.8	64.1	6.6	60.2	5.9	8.3	4.4
2	2	629.4	115.8	758.5	80.1	70.9	8.7	58.5	5.1	8.2	3.2
2	3	717.0	218.2	728.2	61.9	75.7	11.4	59.1	6.8	8.8	4.5
2	4	674.1	200.5	695.8	75.7	76.4	11.9	59.2	6.5	7.0	4.3

APPENDIX V (Continued) - GROUP 2 (Elderly)

Endogenous Target Paradigm

		RT		MT		PV		% Dec		Error	
Cue	Target	M	SD	M	SD	M	SD	M	SD	M	SD
3	1	649.8	140.5	786.3	107.8	63.7	9.4	60.9	5.6	8.1	3.5
3	2	674.5	162.2	778.1	144.5	70.9	11.1	58.9	6.0	8.8	3.0
3	3	639.4	112.6	726.4	78.9	78.0	10.6	60.0	4.4	8.2	2.6
3	4	657.0	130.7	702.1	103.5	74.9	14.0	58.2	6.7	9.9	4.5
4	1	647.5	131.8	763.8	87.8	64.0	6.8	57.3	5.9	7.9	3.0
4	2	687.1	180.4	766.5	81.9	71.1	7.6	61.8	6.6	7.8	3.1
4	3	667.9	145.3	714.0	107.6	79.2	13.3	60.5	4.8	7.2	3.7
4	4	599.3	119.6	696.2	85.5	76.7	11.1	60.3	4.1	8.7	3.3

APPENDIX V (Continued) - GROUP 3 [AD(A)]

Exogenous Target Paradigm

		RT		MT		PV		% Dec		Error	
Cue	Target	M	SD	M	SD	M	SD	M	SD	M	SD
0	1	911.8	348.9	815.8	94.4	62.9	10.1	60.7	4.8	8.4	2.9
0	2	870.3	296.7	783.7	101.7	67.6	10.2	60.8	5.1	7.4	2.5
0	3	930.7	446.2	754.3	80.8	70.9	7.9	60.3	4.7	8.5	2.9
0	4	921.2	355.7	741.1	104.3	68.8	8.2	65.0	3.6	7.5	3.2
1	1	685.5	295.5	801.7	84.6	63.1	11.9	59.3	3.1	7.6	2.9
1	2	803.5	268.1	759.7	134.2	68.1	11.9	57.3	6.4	8.4	4.5
1	3	898.5	297.6	817.2	188.2	67.9	13.4	63.6	5.6	8.3	3.4
1	4	1193.4	556.7	783.6	125.3	66.8	9.7	65.6	5.1	8.5	2.0
2	1	929.8	441.6	822.7	98.4	63.7	13.3	58.8	4.9	6.8	3.2
2	2	667.8	260.9	772.5	96.3	68.1	11.2	58.7	3.2	8.0	3.2
2	3	794.1	294.8	748.9	82.4	72.6	8.9	61.1	5.6	8.6	4.5

APPENDIX V (Continued) - GROUP 3 [AD(A)]

Exogenous Target Paradigm

		RT		MT		PV		% Dec		Error	
Cue	Target	M	SD	M	SD	M	SD	M	SD	M	SD
2	4	806.1	206.7	750.5	119.5	67.9	11.7	65.6	6.3	7.0	2.2
3	1	829.6	298.1	800.4	73.4	64.4	9.6	56.4	7.4	7.4	3.5
3	2	785.2	277.8	825.4	123.9	66.2	13.9	60.8	5.7	8.9	5.6
3	3	614.0	248.7	750.3	88.2	70.6	9.0	61.5	4.2	8.8	2.9
3	4	701.9	289.9	753.2	141.0	68.8	10.1	64.8	8.3	9.1	4.8
4	1	1019.2	517.3	771.5	107.4	65.3	13.2	60.9	8.8	7.5	4.2
4	2	994.5	738.5	777.1	125.2	68.5	14.3	58.9	7.1	8.9	3.3
4	3	845.2	428.8	758.3	118.5	70.8	11.6	58.4	6.8	8.1	3.1
4	4	656.8	256.6	730.1	88.1	68.4	7.4	63.4	3.1	8.5	2.7

APPENDIX V (Continued) - GROUP 3 [AD(A)]

Endogenous Target Paradigm

		RT		MT		PV		% Dec		Error	
Cue	Target	M	SD	M	SD	M	SD	M	SD	M	SD
0	1	1266.1	640.9	849.6	97.9	60.9	9.8	60.1	4.6	7.4	2.9
0	2	1311.5	774.7	817.0	115.1	63.7	7.2	58.8	5.4	6.9	2.4
0	3	1132.6	517.0	813.1	103.3	66.3	7.9	62.2	4.5	8.1	1.9
0	4	1231.1	626.8	789.2	109.6	64.9	7.9	64.5	5.2	8.7	2.3
1	1	956.4	515.5	820.6	111.0	62.3	9.8	59.1	4.2	7.1	1.8
1	2	1123.6	551.4	1011.4	348.5	62.9	9.7	62.0	5.1	8.4	4.4
1	3	1081.0	539.7	1020.8	313.1	64.2	7.3	58.9	6.5	7.1	4.2
1	4	1530.1	761.7	1035.1	505.3	63.4	10.0	64.0	13.3	6.6	3.0
2	1	1295.1	801.7	951.5	280.8	62.6	9.4	62.4	7.3	9.3	4.8
2	2	1005.4	625.4	802.4	92.0	66.1	9.0	57.7	3.8	7.0	2.4
2	3	1199.9	624.9	800.5	79.5	64.8	6.6	58.7	7.6	8.7	2.1
2	4	1056.7	422.6	741.6	111.5	67.3	9.6	65.5	4.9	8.1	3.3

APPENDIX V (Continued) - GROUP 3 [AD(A)]

Endogenous Target Paradigm

		RT		MT		PV		% Dec		Error	
Cue	Target	M	SD	M	SD	M	SD	M	SD	M	SD
3	1	1309.7	685.4	913.4	172.6	64.4	12.5	56.1	7.4	7.3	3.0
3	2	1261.6	834.6	897.5	200.1	63.2	11.2	62.3	7.0	6.8	4.4
3	3	1017.9	481.5	764.7	77.6	67.8	7.9	61.6	2.4	8.9	2.6
3	4	1160.4	495.0	803.5	232.5	66.2	8.8	64.8	5.4	7.9	4.1
4	1	1098.2	651.4	959.7	439.2	62.5	13.7	58.7	7.3	9.1	2.9
4	2	1073.8	443.1	973.4	276.5	62.8	11.4	62.9	10.4	5.6	3.9
4	3	1169.7	757.2	809.8	117.1	67.3	12.0	65.1	5.1	7.5	1.6
4	4	907.9	430.8	769.7	120.7	68.7	7.9	64.2	4.1	8.1	2.7

APPENDIX V (Continued) - GROUP 3 [AD(B)]

Exogenous Target Paradigm

		RT		MT		PV		% Dec		Error	
Cue	Target	M	SD	M	SD	M	SD	M	SD	M	SD
0	1	1480.8	599.0	964.8	157.4	50.4	12.3	62.8	5.8	6.3	9.1
0	2	930.4	354.4	930.4	111.3	57.4	4.2	69.5	4.2	7.8	2.4
0	3	1565.2	184.2	924.5	157.3	55.6	4.5	62.3	6.2	9.1	3.9
0	4	1643.7	521.9	784.1	79.6	59.8	3.8	68.1	7.0	8.3	2.1
1	1	1392.7	323.6	915.3	106.3	50.7	5.2	61.7	1.6	6.5	2.3
1	2	1215.1	566.8	965.3	187.7	53.5	12.4	61.6	9.1	7.6	3.4
1	3	2232.8	693.2	856.6	131.3	59.8	19.8	67.1	4.9	11.5	6.1
1	4	1478.8	403.9	1069.5	329.5	60.0	3.7	77.6	8.7	6.7	4.0
2	1	1512.4	873.6	949.3	212.4	52.3	15.6	67.7	3.3	10.5	11.6

APPENDIX V (Continued) - GROUP 3 [AD(B)]

Exogenous Target Paradigm

		RT		MT		PV		% Dec		Error	
Cue	Target	M	SD	M	SD	M	SD	M	SD	M	SD
2	2	1185.6	621.1	907.0	76.1	52.6	7.6	62.5	3.5	7.7	2.5
2	3	1217.0	947.1	1289.2	996.4	46.8	12.3	46.4	27.7	8.7	3.8
2	4	1234.6	643.9	1097.5	400.3	53.4	5.0	54.4	18.3	6.8	4.0
3	1	933.8	355.4	1020.6	96.3	51.3	4.6	67.8	9.4	8.0	4.0
3	2	1694.1	881.6	891.1	101.8	53.3	7.9	70.6	13.2	8.8	3.0
3	3	1222.7	626.3	759.6	21.9	59.0	6.9	62.7	5.0	7.5	1.3
3	4	2676.2	748.2	936.2	200.8	53.5	3.2	51.6	19.3	9.0	1.0
4	1	1784.3	926.8	944.1	80.2	51.0	4.9	60.5	11.7	7.3	2.6
4	2	1489.3	496.7	920.4	102.1	53.4	10.6	63.1	5.3	6.6	2.8
4	3	1823.5	1261.4	925.9	19.0	52.3	3.3	70.2	8.2	8.3	2.1
4	4	1349.2	230.5	868.7	74.9	57.1	3.9	68.6	6.8	8.1	1.9

APPENDIX V (Continued) - GROUP 4 (PD)

Exogenous Target Paradigm

		RT		MT		PV		% Dec		Error	
Cue	Target	M	SD	M	SD	M	SD	M	SD	M	SD
0	1	689.6	280.8	794.0	128.7	63.2	11.7	56.6	5.3	10.1	4.6
0	2	656.9	219.6	815.2	108.9	64.3	13.8	53.4	7.4	9.0	3.9
0	3	761.0	215.6	776.9	88.4	67.4	14.5	61.4	4.0	8.8	3.4
0	4	734.6	266.8	802.6	156.6	68.2	13.9	59.8	8.9	11.3	5.4
1	1	593.1	244.4	784.9	91.6	62.9	12.3	56.3	4.7	9.9	3.8
1	2	518.2	132.7	795.9	116.1	65.6	14.2	56.9	6.4	8.9	5.2

GROUP 4 (PD)

Exogenous Target Paradigm

		RT		MT		PV		% Dec		Error	
Cue	Target	M	SD	M	SD	M	SD	M	SD	M	SD
1	3	647.5	331.6	722.5	127.8	66.1	18.9	63.6	5.9	9.3	5.5
1	4	792.4	443.9	764.2	128.6	67.2	16.1	59.4	6.1	9.2	4.3

APPENDIX V (Continued) - GROUP 4 (PD)

Exogenous Target Paradigm

		RT		MT		PV		% Dec		Error	
Cue	Target	M	SD	M	SD	M	SD	M	SD	M	SD
2	1	716.8	280.1	790.4	116.8	65.2	15.3	59.2	7.2	10.6	5.7
2	2	560.8	187.9	758.7	94.9	65.3	13.7	59.4	5.8	10.5	5.1
2	3	700.1	484.1	769.1	142.4	64.9	17.1	63.1	6.3	10.2	6.7
2	4	675.9	279.9	730.9	93.8	71.1	15.2	64.6	9.1	9.6	3.3
3	1	891.9	662.5	835.8	126.8	66.2	16.9	57.4	8.7	9.6	7.6
3	2	620.9	225.3	858.7	221.4	64.4	19.7	60.6	7.8	8.1	7.2
3	3	588.2	244.5	750.4	74.6	67.8	13.5	60.2	5.6	9.0	3.8
3	4	648.3	262.8	788.5	137.7	67.5	14.4	61.5	8.3	11.0	6.4
4	1	657.9	420.6	812.5	132.7	62.5	15.3	56.9	7.8	8.0	5.8
4	2	604.4	230.5	763.8	111.6	63.4	13.7	59.4	10.1	11.0	5.9
4	3	614.6	298.8	767.5	111.3	70.4	14.8	61.6	6.9	7.2	3.9
4	4	582.9	247.3	724.3	71.7	67.3	12.0	61.9	4.7	10.9	3.9

APPENDIX V (Continued) - GROUP 4 (PD)

Endogenous Target Paradigm

		RT		MT		PV		% Dec		Error	
Cue	Target	M	SD	M	SD	M	SD	M	SD	M	SD
0	1	963.2	420.8	846.8	181.5	59.1	13.2	56.3	7.1	9.9	2.8
0	2	996.0	589.2	803.4	118.5	62.4	13.8	56.1	8.5	8.5	2.4
0	3	1022.0	673.9	835.3	151.5	61.7	18.1	60.2	5.9	8.8	2.0
0	4	874.8	547.6	778.0	106.8	61.8	14.2	58.4	8.4	10.3	5.5
1	1	808.7	562.4	796.9	116.4	61.0	12.4	55.6	6.4	10.0	3.4
1	2	787.4	292.6	807.9	159.6	59.2	15.3	53.7	7.1	10.3	5.1
1	3	892.7	493.7	824.4	166.6	61.9	17.8	59.6	4.9	9.8	4.8
1	4	900.7	397.3	833.3	154.5	65.9	15.8	65.5	8.1	11.2	6.9
2	1	831.4	347.6	871.5	158.1	58.1	15.0	59.6	12.2	9.5	3.9
2	2	815.4	487.3	779.3	106.3	61.9	14.2	57.1	5.7	10.6	4.5
2	3	773.3	319.2	808.4	147.6	62.3	18.7	57.9	6.3	10.9	7.0
2	4	808.3	478.6	794.5	184.7	63.7	15.4	55.5	8.1	9.8	6.1

APPENDIX V (Continued) - GROUP 4 (PD)

Endogenous Target Paradigm

		RT		MT		PV		% Dec		Error	
Cue	Target	M	SD	M	SD	M	SD	M	SD	M	SD
3	1	783.3	421.6	832.2	162.4	63.1	13.9	58.8	7.9	12.0	8.4
3	2	839.0	477.5	793.3	143.0	61.4	15.1	54.4	6.6	11.0	4.7
3	3	808.1	495.8	774.1	97.2	63.7	16.1	58.8	5.6	10.1	3.8
3	4	835.5	674.7	784.6	171.2	64.7	14.7	60.3	11.0	11.4	6.3
4	1	788.1	468.2	831.8	163.5	62.4	13.5	55.4	7.8	9.7	3.9
4	2	880.1	736.4	868.5	126.2	64.3	15.1	58.9	10.3	9.3	3.1
4	3	728.4	281.5	860.9	245.8	64.6	18.9	59.0	8.8	9.8	6.5
4	4	757.8	386.6	764.8	93.2	65.4	12.4	61.1	6.9	10.7	4.4

References

Abrams, R. A. (1992). Coordination of eye and hand for aimed limb movements. In L. Proteau and D. Elliott (Eds.), Advances in Psychology, 85: Vision and Motor Control. North Holland: Elseiver Science Publishers.

Agid, Y., Ruberg, M., Javoy-Agid, Hirsch, E., Raisman-Vozari, R., Vyas, S., Faucheux, B., Michael, P., Kastner, A., Blanchard, V., Damier, P., Villares, J., & Zhang, P. (1993). Are dopaminergic neurons selectively vulnerable to Parkinson's disease? In H. Narabayashi, T. Nagatsu, N. Yanagisawa, & Y. Mizuno (Eds.), Advances in Neurology, 60. New York: Raven Press, pp. 148-164.

Ainsen, P. S. & Davis, K. L. (1997). The search for disease-modifying treatment for Alzheimer's disease. Neurology, 48(suppl 6), S35-S41.

American Psychiatric Association. (1994). Diagnostic and statistical manual of mental disorders (4th ed.). Washington, DC: Author.

American Psychiatric Association. (1987). Diagnostic and statistical manual of mental disorders (3rd ed. rev). Washington, DC: Author.

Amrhein, P. C., Stelmach, G.E., & Goggin, N. L. (1991). Age differences in the maintenance and restructuring of move ment preparation. Psychology and Aging, 6(3), 451-466.

Amrhein, P. C., von Dras, D., & Anderson, M. (1993). Evidence of direction loss in elderly movement preparation is not due to spatial orienting effects. Experimental Aging Research, 19, 71-95.

Andersen, R. A. (1995). Coordinate transformations and motor planning in posterior parietal cortex. In Michael Gazzaniga (Ed.), The Cognitive Neurosciences, Cambridge, Massachussettes: MIT Press, pp. 519-532.

Andersen, R. A. (1993). Sensory-motor integration and the posterior parietal cortex. In B. Albowitz, K. Albus, U. Kuhnt, H. Northdruft, & P. Wahle (Eds.), Experimental Brain Research, Series 24: Structural and Functional Organization of the Neocortex. New York: Springer Verlag. pp. 344-357.

Andersen, R. A. (1989). Visual and eye movement functions of the posterior parietal cortex. Annual Review of Neuroscience, 12, 377-403.

Andersen, R. A. (1988). The neurobiological basis of spatial cognition: Role of the parietal lobe. In J. Stiles-Davis, M Kritchevsky, and U. Bellugi (Eds.), Spatial Cognition: Brain Bases and Development. New Jersey: Lawrence Erlbaum Associates.

Arbib, M. A. (1985). Schemas for the temporal organization of behavior. Human Neurobiology, 4, 63-72.

Barbosa, E. R., Limongi, J. C. P. & Cummings, J. L. (1997). Parkinson's Disease. The Psychiatric Clinics of North America, 20(4), 769-790.

Barr, W. B., Bilder, R. M., & Kaplan, E. (1990). Patho-physiologic mechanisms underlying spatial disorientation in patients with Alzheimer's disease. Archives of Neurology, 47, 618-619.

Bayles, K. A., Kaszniak, A. W. & Tomoeda, C. K. (1987). Communication and cognition in normal aging and dementia. Boston: College-Hill Publication, Brown & Company.

Bellgrove, M. A., Phillips, J. G., Bradshaw, J. L., Hall, K. A., Presnell, I., & Hecht, H. (1997). Response programming in dementia of the Alzheimer type: A kinematic analysis. Neuropsychologica, 35(3), 229-240.

Benecke, R., Rothwell, J. C, Dick, J. P. R., Day, I, & Marsden, C. D (1986). Performance of simultaneous movements in patients with PD. Brain, 109, 739-757.

Benecke, R., Rothwell, J. C, Dick, J. P. R., Day, I, & Marsden, C. D. (1987). Disturbances of sequential movements in patients with PD. Brain, 110, 361-371.

Benke, T. (1993). Two forms of apraxia in Alzheimer's disease. Cortex, 29, 715-725.

Bennett, K. M. B. & Castiello, U., (1994). Reach to grasp: Changes with age. Journal of Gerontology: Psychological Sciences, 49(1), 1-7.

Bennett, K. M. B., Waterman, C., Scarpa, M. & Castiello, U. (1995). Covert spatial attentional mechanisms in Parkinson's disease. Brain, 118, 153-166.

Beversdorf, D. Q., & Heilman, K. M. (1998). Facilitory paratonia and frontal lobe functioning. Neurology, 51, 968-971.

Blacker, D., Albert, M. S., Bassett, S. S., Go, C. P., Harrell, L. E., & Folstein, M. F. (1994). Reliability and validity of the NINCDS-ADRDA criteria for Alzheimer's disease. Archives of Neurology, 51, 1198-1204.

Bloxham, C. A., Mindle, T. A., & Firth, C. D. (1984). Initiation and execution of predictable and unpredictable movements in Parkinson's disease. Brain, 107, 371-384.

Bock, O., & Arnold, K. (1992). Motor control prior to movement onset: preparatory mechanisms for pointing at visual targets. Experimental Brain Research, 90, 209-216.

Boller, F., Passafiume, P., Keefe, N. C., Rogers, K., Morrow, L., & Kim, Y. (1984). Visuospatial impairment in Parkinson's disease: Role of perceptual and motor factors. Archives of Neurology, 41, 485-490.

Bonnett, M., Requin, J., & Stelmach, G. E. (1991). Changes in electromyographic responses to muscle stretch related to the programming of movement parameters. Electroencephalography and Clinical Neurophysiology, 81, 135-151.

Bradshaw, J. L., & Mattingley, J. B. (1995). Clinical Neuropsychology. New York: Academic Press.

Bradshaw, J. L., Waterfall, M. L., Phillips, J. M., Iansek, R., Mattingley, J. B., & Bradshaw, J. A. (1993). Re-orientation of attention in Parkinson's disease: An extension to the vibrotactile modality. Neuropsychologica, 31(1), 51-66.

Brinkman, J. & Kuypers, H. G. J. M. (1973). Cerebral control of contralateral and ipsilateral arm, hand, and finger movements in the split-brain rhesus monkey. Brain, 96, 653-674.

Brodeur, D. A. & Enns, J. T. (1997). Covert visual orienting across the lifespan. Canadian Journal of Experimental Psychology, 51(1), 20-35.

Brooks, V. B. (1986). The Neural Basis of Motor Control. New York: Oxford University Press.

Brown, S. H. (1996). Control of simple arm movements in the elderly. In A.M. Ferrandez and N. Teasdale (Eds.), Changes in sensory motor behavior in aging. Amsterdam: Elseiver Science Publishers.

Brown, R. G., Jahanshahi, M., & Marsden, C. D. (1993). Response choice in Parkinson's disease. Brain, 116, 869-885.

Brown, R. G., & Marsden, C. D. (1988). Internal vs external cues and the control of attention in Parkinson's disease. Brain, 111, 323-345.

Brown, V. J., Schwartz, U., Bowman, E. M., Fuhr, P., Robinson, D. L., & Hallett, M. (1993). Dopamine dependent reaction time deficits in patients with Parkinson's disease are task specific. Neuropsychologica, 31, 459-469.

Buckolz, E., Hewey, D., Khan, M., & Alain, C. (1994). The influence of attention and response factors upon the spatial precue effect. Human Movement Science, 13, 719-744.

Caffarra, P. Riggio, L., Scaglioni, A., & Freedman, M. (1997). Orienting of visual attention in Alzheimer's Disease: Its implication in favor of hemispheric balance. Neuropsychiatry, Neuropsychology, and Behavioral Neurology, 10(2), 90-95.

Canadian Study of Health and Aging (1994a). The Canadian Study of Heath and aging: Risk factors for Alzheimer's disease in Canada.Neurology, 44, 2073-2080.

Canadian Study of Health and Aging (1994b). The Canadian Study of Heath and aging: Study methods and prevalence of dementia. Canadian Medical Association Journal, 150, 899-913.

Carey, D. P., Hargreaves, E. L., & Goodale, M. A. (1996). Reaching to ipsilateral or contralateral targets: within hemisphere visuomotor processing cannot explain hemispatial differences in motor control. Experimental Brain Research, 112, 496-504.

Carlesimo, G. & Oscar-Berman M. (1992). Memory deficits in Alzheimer's disease: A comprehensive review. Neuropsychology Review, 3(2), 119-169.

Carleton, L. (1992). Visual processing time and the control of movement. In L. Proteau and D. Elliott (Eds.), Advances in Psychology, 85: Vision and Motor Control. North Holland: Elseiver Science Publishers.

Chatterjee, A. (1998). Feeling frontal dysfunction: Facilitory paratonia and the regulation of motor behavior. Neurology, 51, 937-939.

Clark, C. R., Geffen, G. M., & Geffen, L. B. (1987). Catecholamines and attention 1: Animal and clinical studies. Neuroscience and Biobehavioral Reviews, 11, 341-352.

Clark, C. R., Geffen, G. M., & Geffen, L. B. (1989). Catecholamines and the covert orientation of attention in humans. Neuropsychologica, 27(2), 131-139.

Colby, C. L. (1991). The neuroanatomy and neurophysiology of attention. Journal of Child Neurology, 6(Suppl 1991), S90-S118.

Corkin, S. (1981). Acetylcholine, aging and Alzheimer's disease: Implications for treatment. Trends in Neurosciences, 14(2), 287-290.

Cornford, M. E. Chang, L., & Miller, B. L. (1995). The neuropathology of Parkinsonism: An overview. Brain and Cognition, 28, 321-341.

Cossa, F. M., Della Salla, S., & Spinnler, H. (1989). Selective visual attention in Alzheimer's and Parkinson;s Disease: Memory-and data-driven control. Neuropsychologica, 27(6), 887-892.

Cronin-Golomb, A. (1990). Abstract thought in aging and age-related neurological disease. In F. Boller and J. Grafman (Eds.), Handbook of Neuropsychology, Vol 4. Amsterdam: Elseiver Science Publishers.

Crystal, H., Dickson, D., Fuld, P., Masur, D., Scott, R., Mehler, M., Masdeu, J., Kawas, C., Aronson, M., & Wolfson, L. (1988). Clinico-pathologic studies in dementia: Nondemented subjects with pathologically confirmed Alzheimer's disease. Neurology, 38(11), 1682-1687.

Cummings, J. L., & Huber, S. J. (1992). Visuospatial abnormalities in Parkinson's disease. In S.J. Huber and J.L. Cummings (Eds.), Parkinson's Disease: Neurobehavioral Aspects. New York: Oxford University Press.

Cunnington, R., Bradshaw, J. L., & Iansek, R. (1996). The role of the supplementary motor area in the control of voluntary movement. Human Movement Science, 15, 627-647.

D'Aloisio, A. & Klein, R. M. (1990). Aging and the deployment of visual attention. In James T. Enns (Ed.), The Development of Attention: Research and Theory. North Holland: Elseiver Science Publishers.

Darvesh, S. & Freedman, M (1996). Subcortical Dementia: A Neurobehavioral Approach. Brain and Cognition, 31, 230-249.

Deecke, L., (1985). Cerebral potentials related to voluntary actions: PD and normal subjects. In P.J. Delwaide & A. Agnoli (Eds), Clinical Neurophysiology in Parkinsonism. Amsterdam: Elseiver Science Publishers, pp. 91-105.

Dobbs, A. R. (1997a). Evaluations for at-risk experienced drivers. DriveAble Testing Ltd. Edmonton, Alberta

Dobbs, A. R. (1997b). Evaluating the driving competence of dementia patients. Alzheimer Disease and Related Disorders, 11,(Suppl.1), 8-12.

Downing, C. J., & Pinker, S. (1985). The spatial structure of visual attention. In Michael Posner and Oscar Maren (Eds.).Attention and Performance XI, New York: Lawrence Erlbaum Assoc.

Drachman, D. A., & Swearer, J. M. (1993). Driving and Alzheimer's disease: The risk of crashes. Neurology, 43, 2448-2456.

Dubinsky, R. M., Gray, C., Husted, D., Busenbark, K., Vetere-Overfield, B., Wiltfong, D., Parrish, D., & Koller, W. C. (1991), Driving in Parkinson's Disease. Neurology, 41, 517-520.

Duchek, J. M., Hunt, L., Ball, K., Buckles, V., & Morris (1997). The role of selective attention in driving and dementia of the Alzheimer Type. Alzheimer Disease and Associated Disorders, 11(Suppl 1), 48-56.

Egly, R., Driver, J., & Rafal, R. D. (1994). Shifting visual attention between objects and locations: Evidence from normal and parietal lesion patients. Journal of Experimental Psychology: Human Perception and Performance, 123(2), 161-177.

Elliott, D., & Calvert, R. (1990). The influence of uncertainty and premovement visual information on manual aiming. Canadian Journal of Psychology, 44(4), 501-511.

Elliott, D., Carson, R. G., Goodman, D. & Chua, R. (1991). Discrete vs. continuous visual control of manual aiming. Human Movement Science, 10, 393-418.

Feldman, R. S., Meyer, J. S., & Quenzer, L. F. (1997). Principles of Neuropsychopharmacology. Sunderland, Massachusetts: Sinauer Associates Inc. pp. 861-909.

Fisk, J. D., & Doble, S. E. (1992). Cognitive deficits. In George I. Turnbull (Ed.), Physical Therapy Management of Parkinson's Disease. New York: Churchill Livingstone. pp. 69-89.

Fisk, J., & Goodale, M. A., (1984). Differences in the organization of visually guided reaching to ipsilateral and contralateral targets. Behavioral Brain Research, 12, 189-190.

Fisk, J., & Goodale, M. A., (1985). The organization of eye and limb movements during unrestricted reaching to targets in contralateral and ipsilateral visual space. Experimental Brain Research, 60, 159-178.

Fisk, J. D. & Goodale, M. A. (1988). The effects of unilateral brain damage on visually guided reaching: hemispheric differences in the nature of the deficit. Experimental Brain Research, 72, 425-435.

Fisk, J., & Goodale, M. A., (1989). The effects of instructions to subjects on the programming of visually directed reaching movements. Journal of Motor Behavior, 21(1), 5-19.

Folk, C. L. & Hoyer, W. J. (1992). Aging and shifts of visual spatial attention. Psychology and Aging, 7(3), 453-465.

Folstein, M. F., Folstein, S. E., & McHugh, P. R. (1975). "Mini mental state": A practical guide for grading the cognitive state of patients for the clinician. Journal of Psychiatric Research, 12, 189-198.

Foster, N. L., Chase. T. N., Patronas, N. J., Gillespie, M. M. & Fedio, P. F. (1986). Cerebral mapping of apraxia in Alzheimer's disease by positron emission tomography. Annals of Neurology, 19, 139-143.

Friedland, R. P., Koss, E., Kumar, A., Gaine, S., Metzler, D., Haxby, J. V., & Moore, A. (1988). Motor vehicle crashes in dementia of the Alzheimer type. Annals of Neurology, 24, 782-786.

Gawryszewski, L. D. G., Riggio, L., Rizzolatti, G., & Umilta, C. (1987). Movements of attention in the three spatial dimensions and the meaning of "neutral" cues. Neuropsychologica, 25(1), 19-29.

Glencross, D. J. & Barrett, N. (1989). Discrete movements. In Dennis H. Holding (Ed.), Human Skills (2nd ed.). New York: Wiley & Sons. pp. 107-146.

Goggin, N. L., & Stelmach, G. E. (1990). Age-related differences in a kinematic analysis of precued movements. Canadian Journal on Aging, 9(4), 371-385.

Goggin, N. L., & Stelmach, G. E., & Amrhein (1989). Effects of age on motor preparation and restructuring. Bulletin of the Psychonomic Society, 27(3), 199-202.

Goodale, M. A., & Fisk, J. D. (1984). Laterality differences in eye-hand coordination during visually guided reaching. Behavioral Brain Research, 12, 194-195.

Goodale, M. A., Milner, A. D., Jakobson, L. S. & Carey, D. P. (1990). Kinematic analysis of limb movement in neuropsychological research: Subtle deficits and recovery of function. Canadian Journal of Psychology, 44(2), 180-185.

Goodale, M. A., Pelisson, D. & Prablanc, C. (1986). Large adjustments in visually guided reaching do not depend on vision of the hand or perception of target displacement. Nature, 320, 748-750.

Gottsdanker, R. (1980). Aging and the maintaining of preparation. Experimental Aging Research, 6(1), 13-27.

Gottsdanker, R. (1980). Aging and the use of advance probability information. Journal of Motor Behavior, 12(2), 133-143.

Grafton, S. T., Mazziotta, J. C., Woods, R. P., & Phelps, M. E. (1992). Human functional anatomy of visually guided finger movements. Brain, 115, 565-587.

Greenwood, P. M., Parasuraman, R. & Haxby, J. V. (1993). Changes in visuospatial attention over the adult lifespan. Neuropsychologica, 31, 471-485.

Greenwood, P. M., & Parasuraman, R. (1991). Effects of ageing on the speed and attentional cost of cognitive operations. Developmental Neuropsychology, 7(4), 421-434.

Growden, J. H., & Corkin, S. (1986). Cognitive impairments in Parkinson's Disease. In M.D. Yahr & K.J.Bergmann (Eds.), Advances in Neurology, Vol 45: Parkinson's Disease, pp. 383-392.

Haggard, P. & Wing, A. M. (1990). Assessing and reporting the accuracy of position measurements made with optical tracking systems. Journal of Motor Behavior, 22(2), 315-321.

Hart, S. & Semple, J. M. (1990). Neuropsychology and the Dementias. New York: Taylor & Francis.

Hartley, A. A. (1993). Evidence for the selective preservation of spatial selective attention in old age. Psychology and Aging, 8(3), 371-379.

Hartley, A. A., Kieley, J. M. (1995). Adult age differences in the inhibition of return of visual attention. Psychology and Aging, 10(4), 670-683.

Hartley, A. A., Kieley, J. M., & MacKenzie, C. R, M. (1992). Allocation of visual attention in younger and older adults. Perception and Psychophysics, 52(2), 175-185.

Hartley, A. A., Kieley, J. M., & Slabach, E. H. (1990). Age differences and similarities in the effects of cues and prompts. Journal of Experimental Psychology: Human Perception and Performance 16(3), 523-537.

Haxby, J. V., Grady, C. L., Koss, E., Horwitz, B., Schapiro, M. Friedland, R. P., & Rapoport, S. I. (1988). Heterogenous anterior-posterior metabolic patterns in dementia of the Alzheimer type. Neurology, 38, 1853-1863.

Hoehn, M. M. and Yahr, M. D. (1967). Parkinsonism: onset, progression, and mortality. Neurology, 17(5), 427-442.

Hom, J. (1992). General and specific cognitive dysfunctions in patients with Alzheimer's disease. Archives of Clinical Neuropsychology, 7, 121-133.

Huber, S., Shuttleworth, E., & Freidenberg, D. L. (1989). Neuropsychological differences between the dementias of Alzheimer's and Parkinson's diseases. Archives of Neurology, 46(12), 1287-1291.

Hunt, L. A., Murphy, C. F., Carr, D. Duchek, J. M. Buckles, V. & Morris, J. C., (1997). Alzheimer Disease and Associated Disorders, 11(Suppl 1), 13-16.

Inzelberg, R., Flash, T., & Korczyn, A. D. (1990). Kinematic properties of upper limb trajectories in Parkinson's Disease and Idiopathic Torsion Dystonia. In M.B. Streifler, A. D. Korczyn, A. D., E. Melamed, & M. B. H. Youdim (Eds.), Advance in Neurology, Vol 53: Parkinsons Disease: Anatomy, Pathology, and Therapy. pp 183-189, New York: Raven Press.

Iqbal, K. & Wisniewski, H. M. (1983). Neurofibrillary Tangles. In Barry Reisberg (Ed.), Alzheimer's Disease. New York: The Free Press. pp 48-56.

Jahanshahi, M., Brown, R., & Marsden, C. D. (1992). Simple and choice reaction time and the use of advance information for motor preparation in Parkinson's disease. Brain, 115, 539-564.

Jakobson, L. S., & Goodale, M. A. (1991). Factors affecting higher-order movement planning: a kinematic analysis of human prehension. Experimental Brain Research, 86, 199-208.

Jennings, P. J. (1995). Evidence of incomplete motor programming in Parkinson's disease. Journal of Motor Behavior, 27(4), 310-324.

Jones, D. L., Bradshaw, J. L., Phillips, J. G., Iansek, R., Mattingley, J. B., & Bradshaw, J. (1994). Allocation of Attention to programming of movement sequences in Parkinson's disease. Journal of Clinical and Experimental Neuropsychology, 16(1), 117-128.

Jonides, J. (1981). Voluntary versus automatic control over the mind's eye's movement. In J. Long & A. Baddley (Eds.), Attention and Performance IX. New Jersey: Lawrence Erlbaum Publishers.

Jonides, J. & Mack, R. (1984). On the cost and benefit of cost and benefit. Psychological Bulletin, 96(1), 29-44.

Jonides, J., & Yantis, S. (1988). Uniqueness of abrupt visual onset in capturing attention. Perception and Psychophysics, 43(4), 346-354.

Jorm, A. F. (1986). Controlled and automatic information processing in senile dementia: a review. Psychological Medicine, 16, 77-88.

Juola, J. F., Koshino, H., & Warner, C. B. (1995). Tradeoffs between attentional effects of spatial cues and abrupt onsets. Perception and Psychophysics, 57(3), 333-342.

Katzman, R. (1986a). Alzheimer's Disease. Trends in Neurosciences, 9(10), 522-525.

Katzman, R. (1986b). Alzheimer's Disease. The New England Journal of Medicine, 314(15),964-972.

Kelly, J. P. (1991). The neural basis of perception and movement. In E.R. Kandel, J.H. Schwartz and T.M. Jessell (Eds.), Principles of Neural Science. New York: Elseiver.

Kimura, D. (1986). Neuropsychology Test Procedures. London, Ontario: D.K. Consultants.

Kimura, D. (1993). Neuromotor Mechanisms in Human Communication. New York: Oxford University Press.

Klein, R. (1993). On the relationship between overt and covert orienting: A view from human performance. Paper presented at the Third West Coast Attention Meeting, Eugene, Oregon, May, 1993.

Klein, R. (1994). Perceptual-motor expectancies interact with covert visual orienting under conditions of endogenous but not exogenous control. Canadian Journal of Experimental Psychology, 48(2), 167-181.

Klein, R., & Briand, K. (1986). Allocation of attention in visual space. (expanded version of a talk given at the Banff Annual Seminar in Cognitive Science, May, 1986).

Klein, R. & Hansen, E. (1987). Spotlight failure in covert visual attention. Bulletin of the Psychonomic Society, 25(6), 447-450.

Klein, R. & Hansen, E. (1990). Chronometric analysis of apparent spotlight failure in endogenous visual orienting. Journal of Experimental Psychology: Human Perception and Performance, 16(4), 790-801.

Klein, R., Kingstone, A., & Pontefract, A. (1992). Orienting of visual attention. In K. Rayner (Ed.), Eye Movements and Visual Cognition: Scene Perception and Reading.

Klein, R. & Pontefract, A. (1992). Does oculomotor read iness mediate cognitive control of visual attention? In C. Umilta & M. Moscovitch (Eds.), Attention and Performance XV: Conscious and Nonconscious Information Processes. Cambridge, Mass: MIT Press. pp. 333-350.

Kluger, A., Gianutsos, J. G., Golomb, J., Ferris, S. H. George, A. E., Franssen, E., & Reisberg, B. (1997). Patterns of motor impairment in normal aging, mild cognitive decline, and early Alzheimer's disease. Journal of Gerontology: Psychological Sciences, 52B(1), P28-P39.

Komilis, E., Pelisson, D., & Prablanc, C. (1993). Error processing in pointing at randomly feedback-induced double-step stimuli. Journal of Motor Behavior, 25(4), 299-308.

Koshino, H., Warner, B. & Juola, J. F. (1992). Relative effectiveness of central, peripheral, and abrupt-onset cues in visual attention. The Quarterly Journal of Experimental Psyc hology, 45A(4), 609-631.

Kritikos, A., Leahy, C., Bradshaw, J. L., Iansek, R., Phillips, J. G., & Bradshaw, J. A. (1995). Contingent and non-contingent auditory cuing in Parkinson's disease. Neuropsychologica, 33(10), 1193-1202.

Kuljis, R. O. (1994). Lesions of the pulvinar in patients with Alzheimer;s disease. Journal of Neuropathology and Experimental Neurology, 53(2), 202-211.

Lafleche, G., & Albert, M. S., (1995). Executive function in mild Alzheimer's disease. Neuropsychology, 9(3), 313-320.

Lamarre, Y., & Chapman, C. E. (1986). Comparative Timing of neuronal discharge in cortical and cerebellar structures during a simple arm movement in the monkey. In H. Huer and C. Fromm (Eds.), Experimental Brain Research, Series 15: Generation and Modulation of Action Patterns. Springer Verlag.

Larish, D. D. & Frekany, G. A. (1985). Planning and preparing expected and unexpected movements: Reexamining the relationships of arm, direction, and extent of movement. Journal of Motor Behavior, 17(2), 168-189.

Larish, D. & Stelmach, G. (1982). Preprogramming, programming, and reprogramming of aimed hand movements as a function of age. Journal of Motor Behavior, 14(4), 322-340.

La Rue, A. (1992). Aging and Neuropsychological Assessment. New York: Plenum Press.

Lawrence, A. D. & Sahakian, B. J. (1995). Alzheimer Disease, attention, and the cholinergic system. Alzheimer Disease and Associated Disorders, 9(Suppl. 2), 43-49.

Lerner, N. (1994). Giving the older driver enough perception-reaction time. Experimental Aging Research, 20, 25-33.

Levin, B. E., Llabre, M. M., Ansley, J., Weiner, W. J., & Sanchez-Ramos, J. (1990). Do Parkinsonians exhibit visuospatial deficits? In M.B. Srreifler, A.D. Korczyn, E. Melamed, and M. B. H. Youdim (Eds.), Advances in Neurology, 53: Parkinson's Disease: Anatomy, Pathology, and Therapy. New York: Raven Press. pp. 311-315.

Madden, D. J., Nebes, R.D., & Allen, P. A. (1992). Cognitive slowing in Alzheimer's disease as a function of task type and response type. Developmental Neuropsychology, 8(4), 459-471.

Mahurin, R. K., Feher, E. P., Nance, M. L., Levy, J. K., & Pirozzolo, F. J. (1993). Cognition in Parkinson's disease and related disorders. In R.W. Parks, R.F. Zec, and R.S. Wilson (Eds.), Neuropsychology of Alzheimer's Disease and Other Dementias. New York: Oxford University Press.

Majsak, M. J., Kaminski, T., Gentle, M. & Flanagan, R. (1998). The reaching movements of patients with Parkinson's Disease under self-determined maximal speed and visually cued conditions. Brain, 121, 755-766.

Marteniuk, R. G. (1976). Information Processing in Motor Skills. New York: Holt, Rinehart, and Winston.

Marteniuk, R. J., MacKenzie, C. L., Jeannerod, M., Athenes, S., & Dugas, C. (1987). Constraints on human arm movement trajectories. Canadian Journal of Psychology, 41, 365-378.

Martin, O. & Prablanc, C. (1992). Online control of hand reaching at undetected target displacements. In G.E. Stelmach & J. Requin (Eds.), Tutorials in Motor Behavior II. Amsterdam: Elseiver Publishers.

Maruff, P., Malone, V., & Currie, J. (1995). Asymmetries in the covert orienting of visual spatial attention to spatial and non-spatial cues in Alzheimer's disease. Brain, 118, 1421-1435.

McCormick, P. A. (1997). Orienting attention without awareness. Journal of Experimental Psychology: Human Perception and Performance, 23(1), 168-180.

McCormick, P. A., & Klein, R. (1990). The spatial distribution of attention during covert visual orienting. Acta Psychologica, 75, 225-242.

McGeer, E. G., & McGeer, P. L. (1997). Aging, neuro-degenerative disease and the brain. Canadian Journal on Aging, 16(2), 218-236.

McKhann, G., Drachman, D., Folstein, M., Katzman, R., Price, D., & Stadlan, M. (1984). Clinical diagnosis of Alzheimer's Disease: Report of the NINCDS-ADRDA work group under the auspices of Department of Health and Human Services Task Force on Alzheimer's Disease. Neurology, 34, 939-943

Metter, E. J., Kuhl, D. E., & Riege, W. H. (1990). Brain glucose metabolism in Parkinsons disease. In M.B. Streifler, A.D. Korczyn, A. D., E. Melamed, & M. B. H. Youdim (Eds.), Advance in Neurology, Vol 53: Parkinsons Disease: Anatomy, Pathology, and Therapy. New York: Raven Press. pp 135-139.

Molsa, P. K., Sako, L., Rinne, J. O., & Rinne, U. K. (1987). Alzheimer's disease: neuropathological correlates of cognitive and motor disorders. Acta Neurologica Scandinavia, 75, 376-384.

Montgomery, E. B., Gorman, D. S., & Nuessen, J. (1991). Motor initiation versus execution in normal and Parkinson's disease subjects. Neurology, 41, 1469-1475.

Montgomery, E. B., Nuessen, J., & Gorman, D. S. (1991). Reaction time and movement velocity abnormalities in Parkinson's disease under different task conditions. Neurology, 41, 1476-1481.

Morris, J. C. (1997). Forward. Alzheimer Disease and Related Disorders, 11,(Suppl.1), 1-2.

Morris, M. E., & Iansek, R. (1996). Characteristics of motor disturbance in Parkinson's disease and strategies for movement rehabilitation. Human Movement Science, 15, 649-669.

Mortimer, J. A., Pirozzolo, F. J., and Maletta, G. J. (1982). Overview of the aging motor system. In J.A. Mortimer, F.J. Pirozzolo, & G. Maletta (Eds.), The aging motor system, New York: Praeger Scientific. pp 1-6.

Mortimer, J. A., & Webster, D. D. (1982). Comparison of extrapyramidal motor function in normal aging and Parkinson's Disease. In J. A. Mortimer, F. J. Pirozzolo, & G. J. Maletta (Eds.), The Aging Motor System. New York: Praeger. pp. 217-241.

Muller, H. J., & Rabbitt, P. M. A. (1989). Reflexive and voluntary orienting of visual attention: Time course of activation and resistance to interruption. Journal of Experimental Psychology: Human Perception and Performance, 15(2), 315-330.

Muller, F., & Stelmach, G. E. (1992). Prehension movements in Parkinson's disease. In G.E. Stelmach & J. Requin (Eds.), Tutorials in Motor Behavior II. Amsterdam: Elseiver Science Publishers.

Navon, D. (1985). Attention division or attention sharing? In Michael I. Posner and Oscar S. M. Marin (Eds.), Attention and Performance XI. Hillside, New Jersey: Lawrence Erlbaum Associates. pp. 133-146.

Nebes, R. D., & Brady, C. B. (1989). Focused and divided attention in Alzheimer's disease. Cortex,25, 305-315.

Nestor, P. G., Parasuraman, R. & Haxby, J. V. (1991). Speed of information processing and attention in early Alzheimer's dementia. Developmental Neuropsychology, 7(2), 243-256.

Nissen, M. J., & Corkin, S. (1985). Effectiveness of attentional cuing in older and younger adults. Journal of Gerontology, 40(2), 185-191.

Ochipa, C., Gonzalez Rothi, L. J., & Heilman K. M. (1992). Conceptual apraxia in Alzheimer's disease. Brain, 115, 1061-1071.

Oken, B. S., Kishiyama, S. S., Kaye, J. A., & Howieson, D. B. (1994). Attention deficit in Alzheimer's Disease is not simulated by an anticholonergic/antihistaminergic drug and is distinct from deficits in healthy aging. Neurology, 44, 657-662.

Oleske, D. M., Wilson, R. S., Bernard, B. A., Evans, D. A., & Terman, E. W. (1995). Epidemiology of injury in people with Alzheimer's Disease. Journal of the American Geriatric Society, 43, 741-746.

Owsley, C., Ball, K., Sloane, M. E., Roenker, D. L., & Bruni, J. R. (1991). Visual/cognitive correlates of vehicle accidents in older drivers. Psychology and Aging, 6(3), 403-415.

Parasuraman, R. Greenwood, P. M., Haxby, J. V., & Grady, C. L. (1992). Visuospatial attention in dementia of the Alzheimer type. Brain, 115, 711-733.

Parasuraman, R., & Haxby, J. (1993). Attention and Brain function in Alzheimer's disease: a review. Neuropsychology, 7(3), 242-272.

Parks, R. W., Haxby, J. V., & Grady, C. L. (1993). In R. W. Parks, R. F. Zec, & R. S. Wilson (Eds.), Neuropsychology of Alzheimer's Disease and other Dementias. New York: Oxford University Press.

Parasuraman, R., & Nestor, P. G. (1991). Attention and Driving Skills in Aging and Alzheimer's Disease. Human Factors, 33(5), 539-557.

Pelisson, D., Prablanc, C., Goodale, M. & Jeannerod, J. (1986). Visual control of reaching movements without vision to the limb. Experimental Brain Research, 62, 303-311.

Petersen, S. E., Robinson, D. L., & Currie, J. N. (1989). Influences of lesions of parietal cortex on visual spatial attention in humans. Experimental Brain Research, 76, 267-280.

Petersen, S. E., Robinson, D. L., & Morris, J. D. (1987). Contributions of the pulvinar to visual spatial attention. Neuropsychologica, 25(1A), 97-105.

Pirozzolo, F. J., Mahurin, R. K., & Swihart, A. A. (1991). Motor function in aging and neurodegenerative disease. In F. Boller and J. Grafman (Eds.), Handbook of Neuropsychology, 5,, 167-194.

Playfer, J. (1989). Parkinson's Disease and other Parkinsonian syndromes. In R. Tallis (Ed.), The Clinical Neurology of Old Age. New York: John Wiley & Sons. pp. 126-139.

Poizner, L. J., Mack, L., Verfaellie, M., Rothi, L. J. G., & Heilman, K. M. (1990). Three-dimensional computergraphic analysis of apraxia: Neural representations of learned movement. Brain, 113, 85-101.

Posner, M. I. (1980). Orienting of Attention. Quarterly Journal of Experimental Psychology, 32, 3-25.

Posner, M. I. (1986). Orienting. Chronometric Explorations of Mind: The third Paul M. Fitts Lectures. New York: Oxford University Press. pp. 185-216.

Posner, M. I. (1989). Structures and function of selective attention. In Thomas Boll and Brenda Byrant (Eds.), Clinical Neuropsychology and Brain Function: Research, Measurement and Practice. Washington: American Psychological Association.

Posner, M. I. (1995). Attention in cognitive neuroscience: An overview. In Michael S Gazzaniga (Ed.), The Cognitive Neurosciences.Cambridge, Massachussettes: MIT Press. pp. 185-216.

Posner, M. & Cohen, Y. (1980). Attention and the control of movements. In G. E. Stelmach & J Requin (Ed.), Tutorials in Motor Behavior. Amsterdam: North Holland Publ.

Posner, M., Inhoff, A. W., Friedrich, F. J., & Cohen, Y. (1987). Isolating attentional systems: A cognitive-anatomical analysis. Psychobiology, 15(2), 107-121.

Posner, M., Nissen, M. J., & Ogden, W.C. (1978). Attended and unattended processing modes: The role of set for spatial location. In Herbert L. Pick and Elliot Saltzman (Eds.), Modes of Perceiving and Processing Information. New York: Lawrence Erlbaum Assoc.

Posner, M. & Petersen, S. E. (1990). The attention system of the human brain. Annual Review of Neuroscience, 13, 25-42.

Posner, M., Petersen, S. E., Fox, P., & Raichle (1988). Localization of cognitive operations in the human brain. Science, 240, 1627-1631.

Posner, M. & Snyder, C. (1975). Attention and cognitive control. In R.L. Solso (Ed.), Information Processing and Cognition. The Loyola Symposium.

Posner, M. & Snyder, C. R. & Davidson, B. J. (1980). Attention and the detection of signals. Journal of Experimental Psychology: General, 109 (2), 160-174.

Posner, M., Walker, J. A. Friedrich, F. J., & Rafal, R. D. (1984). Effects of parietal injury on covert orienting of attention. The Journal of Neuroscience, 4(7), 1863-1874.

Posner, M., Walker, J. A. Friedrich, F. J., & Rafal, R., D. (1987). How do the parietal lobes direct covert attention? Neuropsychologica, 25 (1A), 135-145.

Praamstra, P., Meyer, A.S., Cools, A. R., Horstink, M. W. I. M., & Stegeman, D. F. (1996). Movement preparation in Parkinson's disease: Time course and distribution of movement-related potentials in a movement precuing task. Brain, 119, 1689-1704.

Prablanc, C. & Martin, O. (1992). Automatic control during hand reaching at undetected two-dimensional target displacements. Journal of Neurophysiology, 67(2), 455-469.

Prablanc, C., Pelisson, D., & Goodale, M. A. (1986). Visual control of reaching movements without vision of the limb. Experimental Brain Research, 62, 293-302.

Pratt, J., Chasteen, A. L., & Abrams, R. A. (1994). Rapid aimed limb movements: Age differences and practice effects in component submovements. Psychology and Aging, 9(2), 325-334.

Rafal, R. D., Calabresi, P. A., Brennan, C. W., & Sciolto (1989). Saccade preparation inhibits reorienting to recently attended locations. Journal of Experimental Psychology: Human Perception and Performance, 15(4), 673-685.

Rafal. R. D., & Posner, M. I. (1987). Deficits in human visual spatial attention following thalamic lesions. Proceeding of the National Academy of Sciences, 84, 7349-7353.

Rafal, R. D. Posner. M. I., Friedman, J. H., Inhoff, A. W., & Bernstein, E. (1988). Orienting of visual attention in Progressive Nuclear Palsy. Brain, 111, 267-280.

Rafal, R. D., Posner, M. I., Walker, J. A. & Friedrich, F. J. (1984). Cognition and the basal ganglia: Separating mental and motor components of performance in Parkinson's disease. Brain, 107, 1083-1094.

Rapcsak, S. Z., Croswell, S. C., & Reubens, A. B. (1989). Apraxia in Alzheimer's disease. Neurology, 39, 664-668.

Requin, J. (1992). From action representation to movement control. In G.E. Stelmach & J. Requin (Eds.), Tutorials in Motor Behavior II. Amsterdam: Elseiver Science Publ. pp 159-179.

Reinanch, S. J., Rizzo, M., & McGehee, D. V. (1997). Driving with Alzheimer Disease: The anatomy of a crash. Alzheimer Disease and Related Disorders, 11(Suppl.1), 21-27.

Reuter-Lorenz, P. A., & Fendrich, R. (1992). Oculomotor readiness and covert orienting: Differences between central and peripheral precues. Perception and Psychophysics, 52(3), 336-344.

Revonsuo, A., Portin, R., Koivikko, L., Rinne, J. O., & Rinne, U. K. (1993). Slowing of information processing in Parkinson's disease. Brain and Cognition, 21, 87-110.

Rhawn, J. (1990). The frontal lobes: Neuropsychiatry, Neuropsychology, and behavioral neurology. In Joseph Rhawn's (Ed.), Neuropsychology, Neuropsychiatry, and Behavioral Neurology. New York: Plenum Press. pp. 139-195.

Richards, M. & Stern, Y. (1992). Cognitive studies of Alzheimer's disease. In D. J. Stein and J. E. Young (Eds.), Cognitive Science and Clinical Disorders. New York: Academic Press Inc. pp. 289-311.

Rizzolatti, G., Riggio, L., Dascola, I., & Umilta, C. (1987). Reorienting attention across the horizontal and vert ical meridians: evidence in favor of a premotor theory of attention. Neuropsychologica, 25(1A), 31-40.

Robin, D. A., & Rizzo, M. (1992). Orienting attention in audition and between audition and vision: Young and elderly subjects. Journal of Speech and Hearing Research, 35, 701-707.

Robinson, D. L., & Petersen, S. E., (1992). The pulvinar and visual salience. Trends in Neurosciences, 15(4), 127-132.

Rosenbaum, D. A. (1980). Human Movement Initiation: Specification of arm, direction, and extent. Journal of Experimental Psychology: General, 109(4), 444-474.

Rosenbaum, D. A. (1983). The movement precuing techniqu e: Assumptions, applications, and extensions.In Richard Magill (Ed.), Memory and the Control of Action. North Holland.

Rosenbaum, D. A., & Kornblum, S. (1982). A priming method for investigating the selection of motor responses. Acta Psychologica, 51, 223-243.

Roy, E. A., Weir, P. L., & Leavitt, J. L. (1996). Constraints on prehension: A framework for studying the effects of aging. In A.M. Ferrandez & N. Teasdale (Eds.), Changes in Sensory Motor Behavior in Aging. New York: Elseiver Science Publ. pp. 279-314.

Roy, E. A., Winchester, T. Weir, P., & Black, S. (1993). Age differences in the control of visually aimed movements. Journal of Human Movement Studies, 24, 71-81.

Sakata, H., Taira, M., Mine, S. & Murata, A. (1992). Hand-movement-related neurons of the posterior parietal cortex of the monkey: Their role in the visual guidance of hand movements.In R. Caminiti, P.B. Johnson, and Y. Burnod (Eds.), Control of Arm Movements in Space: Neurophysiological and Computational Approaches. New York: Springer-Verlag. pp 185-198.

Salthouse, T. A. (1985). Speed of behavior and its impl ications for cognition. In James E. Biren and K. Warner Schaie (Eds.), Handbook of the Psychology of Aging, 2nd edition. New York: Van Nostrand Reinhold Co.

Sanes J. N. & Evarts, E. V. (1984). Motor Psychophysics. Human Neurobiology, 2, 217-225.

SAS (1982). Statistical Analysis Institute Inc: Carey N.C.

Schofield, P. W., Tang, M., Marder, K., Bell, K., Dooneief, G., Lantigua, R., Wilder, D., Gurland, B., Stern. Y., & Mayeux, R. (1995). Consistency of clinical diagnosis in a community-based longitudinal study of dementia and Alzheimer's disease. Neurology, 45, 2159-2164.

Schmidt, R. A. (1975). Motor Skills. New York: Harper and Rowe Publishers.

Schneider, W., Dumais, S. T., & Shriffrin, R. M. (1984). Automatic and control processing and attention. In R. Parasuraman and D. Davies (Eds.), Varieties of Attention. New York: Academic Press.

Scinto, L. F. M., Daffner, K. R., Castro, L., Weintraub, S., Vavrik, M. & Mesulam, M. M. (1994). Impairment of spatially directed attention in patients with probable Alzheimer's disease as measured by eye movements. Archives of Neurology, 51, 682-688.

Semjen, A., & Gottsdanker, R. (1992). Plans and programs for short movement sequences. In G.E. Stelmach and J. Requin (Eds.), Tutorials in Motor Behavior II. Amsterdam: Elseiver Science Publishers.

Seshardri, S., Drachman, D. A., & Lippa, C. F. (1995). Apopipoprotein E e4 Allele and the lifetime risk of Alzheimer's disease. Archives of Neurology, 52, 1074-1079.

Shaffer, L. H. (1992).Motor programming and control. In G. E. Stelmach & J. Requin (Eds.), Tutorials in Motor Behavior II. Amsterdam: Elseiver Science Publ. pp 181-194.

Sharp M. H. (1990). Patients with early Parkinson's disease are not impaired on spatial orienting of attention. Cortex, 26, 515-524.

Sheliga, B. M., Craighero, L., Riggio, G., & Rizzolatti, G. (1997). Effects of spatial attention on directional manual and ocular responses. Experimental Brain Research, 114, 339-351.

Shepherd, M., & Muller, H. J. (1989). Movement versus focusing of visual attention. Perception and Psychophysics, 46(2), 146-154.

Sheridan, M. R., Flowers, K. A., & Hurrell, J. (1987). Programming and execution of movement in Parkinson's disease. Brain, 110, 1247-1271.

Sims, N. R. & Bowen, D. M. (1983). Changes in choline acetytransferase and in acetylcholine synthesis. In Barry Reisberg (Ed.), Alzheimer's Disease. New York: The Free Press. pp 88-92.

Smith, G. A., & Brewer, N. (1995). Slowness and age: Speed-accuracy mechanisms. Psychology and Aging, 10(2), 238-247.

Spinnler, H. (1991). The role of attention disorders in the cognitive deficits of dementia. In F. Boller and J. Grafman (Eds.), Handbook of Neuropsychology, 5, 79-122.

Spirduso, W. W. & MacRae, G. (1990). Motor performance and aging. In J. E. Birren & W. Schaie (Eds.), Handbook of The Psychology of Aging (3rd ed.). New York: Academic Press Inc.

Stacy, M., & Jankovic, J. (1992). Clinical and neurobiological aspects of Parkinson's disease. In S.J. Huber and J.L. Cummings (Eds.), Parkinson's Disease: Neurobehavioral Aspects. New York: Oxford University Press. pp. 10-31.

Stelmach, G. E., Amrhein, P. C. & Goggin, N. L. (1988). Age differences in bimanual coordination. Journal of Gerontology: Psychological Sciences, 41(1), 18-23.

Stelmach, G. E., Goggin, N. L., & Amrhein, P. C. (1988). Aging and the restructuring of precued movements. Psychology and Aging, 3(2), 151-157.

Stelmach, G. E., Goggin, N. L., & Garcia-Colera, A. (1987). Movement specification time with age. Experimental Aging Research, 13(1), 39-46.

Stelmach, G. E., Worrington, C. J., & Strand, E. A. (1986). Movement preparation in Parkinson's disease: The use of advance information. Brain, 109, 179-1194.

Stuss, D. T., Shallice, T. Alexander, M. P. & Picton, T. W. (1995). A multidisciplinary approach to anterior attentional functions. Annals of the New York Academy of Sciences, 769, 191-211.

Summers, J. J. (1989). Motor Programs. In D. H. Holding (Ed.), Human Skills, 2nd Edition. New York: John Wiley and Sons.

Tabachnick B. G., & Fidell, L. S., (1989). Using Multivariate Statistics. New York: Harper & Row.

Taira, M., Mine, S., Georgopoulos, A. P., Murata, A., & Sakata, H. (1990). Parietal cortex neurons of the monkey related to the visual guidance of hand movements. Experimental Brain Research, 83, 29-36.

Taylor, A. E., & Saint-Cyr (1992). Executive function. In S. J. Huber and J. L. Cummings (Eds.), Parkinson's Disease: Neurobehavioral Aspects. New York: Oxford University Press. pp 74-85.

Tellinghuisen, D. J., Zimba, L. D., & Robin, D. A. (1996). Endogenous visuospatial precuing effects as a function of age and task demands. Perception and Psychophysics, 58(6), 947-958.

Todor, J. I. & Smiley, A. L. (1985). Performance differences between the hands: Implications for studying disruption to limb praxis. In J. I. Tudor and A. L. Smiley's (Eds.), Neuropsychological Studies of Apraxia and Related Disorders. North Holland: Elseiver Science Publ. 309-343.

Tuokko, H., Kristjansson, E., & Miller, J. (1995). Neuropsychological detection of dementia: An overview of the neuropsychological component of the Canadian study of health and aging. Journal of Clinical and Experimental Neuropsychology, 17(3), 352-373.

Umilta, C. (1988). Orienting of attention. In F. Boller & J. Grafman (Eds.), Handbook of Neuropsychology, Vol 1, North Holland: Elsevier Science Publishers, B.V.

Umilta, C., Riggio, L., Dascola, I., & Rizzolatti, G. (1991). Differential effects of central and peripheral cues on the reorienting of spatial attention. European Journal of Cognitive Psychology, 3(2), 247-267.

Voytko, M. L., Olton, D. S., Richardson, R. T., Gorman, L. K., Tobin, J. R., & Price, L. (1994). Basal Forebrain Lesions in monkeys disrupt attention but not learning and memory. The Journal Of Neuroscience, 14(1), 167-186.

Walker, N. Philbin, D. A., & Fisk, A. D. (1997). Age-related differences in movement control: Adjusting submovement structure to optimize performance. Journal of Gerontology: Psychological Sciences, 52B(1), 40-52.

Warabi, T., Noda, H., & Kato, T. (1986). Effect of aging on sensorimotor functions of eye and hand movements. Experimental Neurology, 92, 686-697.

Welford, A. T. (1982). Motor skills and aging. In J. A. Mortimer, F. J. Pirozzolo, & G. J. Maletta (Eds.), The Aging Motor System. New York: Praeger Publishers.

Welford, A. T. (1988). Reaction time, speed of performance, and age. Annals of the New York Academy of Sciences, 515, 1-17.

White, R., Au, R., Durso, R, & Moss, M. B. (1992). Neuropsychological function in Parkinson's disease. In R. F. White (Ed.), Clinical Syndromes in Adult Neuropsychology: The Practioner's Handbook. Elseiver Science Publishers, Amsterdam.

Whitehouse, P. J., Price, D. L., Clark, A. W., Coyle, J. T., & Delong, M. R. (1981). ALzheimer's disease: Evidence for selective loss of cholinergic neurons in the nucleus basalis. Annals of Neurology, 10(2), 122-126

Wichman, T., & Delong, M. R. (1993). Pathophysiology of Parkinsonian motor abnormalities. In H. Narabayashi, T. Nagatsu, N. Yanagisawa, & Y. Mizuno (Eds.), Advances in Neurology, 60. New York: Raven Press, pp. 53-61.

Wisniewski, H. M. (1983). Neuritic (Senile) and Amyloid Plaques. In Barry Reisberg (Ed.), Alzheimer's Disease. New York: The Free Press. pp 57-61.

Wright, M. J., Burns, R. J., Geffen, G. M., & Geffen, L. B. (1990). Covert orientation of visual attention in Parkinson's disease: An impairment in the maintenance of attention. Neuropsychologica, 28(2), 151-159.

Wright, M. J., Cremona-Meteyard, S. L. Geffen, L. B., & Geffen, G. M. (1994). The effects of closed head injury, senile dementia of the Alzheimer's type, and Parkinson's Disease on covert orientation of attention. Australian Journal of Psychology, 46(2),63-72.

Wright, M. J., Geffen, G. M., & Geffen, L. B. (1993). Event-related potentials associated with covert orientation of visual attention in Parkinson's disease. Neuropsychologica, 31(12), 1283-1297.

Wright, R. D., Richard, C. M., & McDonald, J. J. (1995). Neutral location cues and cost/benefit analysis of visual attention shifts. Canadian Journal of Experimental Psychology, 49(4), 540-548.

Wright, R. D. & Ward, L. M. (1994). Shift of visual attention: An historical and methodological overview. Canadian Journal of Experimental Psychology, 48(2), 151-166.

Yamada, T., Izyuuinn, M, Schulzer, M., & Hirayama, K. (1990). Covert orienting attention in Parkinson's disease. Journal of Neurology, Neurosurgery, and Psychiatry, 53, 593-596.

Yanagisawa, N., Fujimoto, S., & Tamaru, F. (1989). European Neurology, 29(Suppl 1), 19-28.

Yantis, S. & Jonides, J. (1984). Abrupt visual onsets and selective attention: Evidence from visual search. Journal of Experimental Psychology: Human Perception and Performance, 10(5), 601-621.

Yoshida, M. (1993). The neuronal mechanism underlying Parkinsonism and dyskinesia, and differential roles of the putamen and caudate nucleus. In H. Narabayashi, T. Nagatsu, N. Yanagisawa, & Y. Mizuno (Eds.), Advances in Neurology, 60. New York: Raven Press, pp. 71-77.

Zec, R. F. (1993). Neuropsychological functioning in Alzheimer's Disease. In Randolph W. Parks, Ronald F. Zec and Robert S. Wilson (Eds.), Neuropsychology of Alzheimer's Disease and other Dementias. New York: Oxford.